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Personalized Models of Social Anxiety Disorder and Depression

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by
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ABSTRACT OF THE DISSERTATION

Personalized Models of Risk for Social Anxiety Disorder and Depression

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Social anxiety disorder (SAD) is an important risk factor for major depressive disorder (MDD) and together this comorbidity constitutes a highly impairing syndrome and vicious cycle of symptomatology, associated with tremendous health costs and societal burden. Despite much group-level research examining risk factor for MDD specifically, there is limited group and individual-level research evaluating how individuals with SAD transition into depressive episodes. Clinical and theoretical evidence suggests that each patient may exhibit a unique personalized pattern of risk factors. These idiographic patterns may contradict relationships seen at the group level. In this dissertation, women (N = 35) with SAD and a current or past major depressive episode were asked to complete brief surveys of their mood and emotional experience five times a day for a month via a smartphone application. These data were analyzed using idiographic analyses to construct individual-level models of each woman's mood. Additionally, a multilevel model was constructed to determine risk factors for daily levels of sadness on the group level. Overall, results largely supported study hypotheses. Most women's models

demonstrated few statistically significant directed pathways predicting sadness, although the directed pathways that existed were different between women. Additionally, there was minimal overlap between the multilevel model and each individual-level model, providing evidence that relationships reflected in the individual-level models differed from the relationships elucidated at the group-level. Differences between the multilevel and individual-level models highlight the potential integration of idiographic methodology into clinical practice. Furthermore, nuances related to the multilevel methodology used here provide evidence as to how intensive longitudinal data can be used to improve upon group-level models of psychopathology. Implications for the use of intensive longitudinal data and idiographic analyses in clinical assessment and intervention are discussed.

Chapter 1: Literature Review

Social anxiety disorder (SAD) and major depressive disorder (MDD) are, collectively, the most prevalent mental disorders afflicting young adults, leading to a tremendous loss of social and economic potential (Acarturk, de Graaf, van Straten, ten Have, & Cuijpers, 2008; Kessler et al., 2005). Billions of healthcare dollars are spent each year on morbidity associated with these disorders, and even more is estimated to be lost in workplace productivity, constituting an enormous public health burden (Greenberg et al., 2003; Kessler et al., 2011; Kessler, Merikangas, & Wang, 2007). Notably, researchers have long noted that young adults with SAD are especially vulnerable to develop MDD, which often arises earlier and is of greater severity than MDD for individuals without SAD (Dalrymple & Zimmerman, 2011; Kessler, 1997; Pini et al., 1997; Stein et al., 2001).

The Cumulative Interpersonal Risk model developed by Epkins and Heckler (2011) describes a diathesis-stress model of SAD leading to MDD. This model suggests that individuals with SAD may be at especially high risk for developing MDD due to shared genetic, affective, and cognitive factors (Cummings, Caporino, & Kendall, 2014; Epkins & Heckler, 2011). Affective factors, such as high levels of negative affect and low levels of positive affect, coupled with negative self-referential biases and uses of cognitive strategies like post-event processing and rumination put individuals at greater risk of developing internalizing disorders (Cummings et al., 2014; Epkins & Heckler, 2011). Thus, the transition from this risk state into MDD is hypothesized to ensue in vulnerable individuals as the result of maladaptive social processes and stressful interpersonal outcomes, such as withdrawal and social rejection (Starr, Hammen, Connolly, & Brennan, 2014). Peer rejection, peer victimization, social withdrawal, and

loneliness are all implicated in the model and related literature as triggers for SAD and development of MDD (Starr & Davila, 2008; Starr et al., 2014).

Numerous studies focusing on the etiology of and risk factors for SAD and MDD have provided support for theoretical models of comorbidity (Hirshfeld-Becker et al., 2008; Hirshfeld-Becker, 2010; Rapee & Spence, 2004; Rudolph, Flynn, & Abaied, 2008) and several treatment outcome studies have demonstrated that treatment of anxiety via cognitive-behavioral therapy also improves MDD symptoms and decreases recurrence of MDD (Stewart & Chambless, 2009). However, despite decades of research on etiological and risk factors for comorbid SAD and MDD, there is limited research on methods for preventing or effectively treating this comorbidity (Epkins & Heckler, 2011). An additional, and perhaps more pervasive, limitation may be the methodology used in previous research. Clinical psychologists have historically used primarily cross-sectional or longitudinal between-subjects (group-level) designs to identify risk factors for MDD (e.g., Alloy et al., 2000), but use of these designs limits researchers to group-level inferences. Arguably, findings from group-level studies do not tell the entire story – risk factors identified at the group-level may be non-significant, or even protective, for specific individuals within the group (Fisher, Medaglia, & Jeronimus, 2018; Molenaar, 2004).

This concept is best illustrated with an example. Researchers may wish to identify risk factors for future depressive symptoms using a between-subjects longitudinal design. Group-level analyses, in which data are aggregated across individuals, suggest that social stress predicts future depressive symptoms. However, in order for researchers to conclude that a specific individual experiences a similar increase in depressive symptoms in reaction to social stress, this individual's data must be consistent with the group-level trends. That is, for any given individual within the group, social stress must predict future depression over time. However, intraindividual

patterns can differ from the group-level trend. For example, one individual's data may show no significant relationship between social stress and future depressive symptoms. Another individual's data may show an inverse relationship – social stress predicts fewer depressive symptoms in the future. Thus, treatment and prevention efforts that are based on group-level risk factors may be limited, as these risk factors may not predict the same outcomes for all, or even most, individuals within the group (Hamaker, 2012; Molenaar, 2004).

The concept of group versus individual differences is addressed in greater depth in Molenaar's discussions of ergodicity – a theoretical property, which refers to the assumption that each part is representative of the whole (Molenaar, 2004). Molenaar and other researchers argue that psychological processes do not meet the assumptions of ergodicity, and thus, group level findings may not accurately represent individual-level relationships (Fisher, Medaglia, & Jeronimus, 2018; Molenaar, 2004). In following, violations to ergodicity allow for the possibility that certain risk factors may carry more predictive value for an outcome for some individuals versus others (Hamaker, 2012). This concept was illustrated in the example above, in which social stress predicted future depressive symptoms for one individual, but did not significantly predict future depressive symptoms for second individual, and predicted fewer depressive symptoms for the third individual. The use of person-specific methodology allows clinical scientists to examine whether risk factors elucidated from group-level designs manifest on the individual-level and if so, how these patterns differ between individuals. Results from idiographic analyses carry useful implications for clinical assessment and intervention. For example, if a clinician knew that maladaptive interpersonal relationships had a stronger impact on his or her client's depression than engagement in (or avoidance) of daily activities, this may assist the therapist in selecting a more targeted empirically supported intervention – in this

example, interpersonal therapy rather than behavioral activation for depression.

Only recently have these fundamental issues been addressed using individual sampling methodology (ISM), which includes the use of within-subjects or person-centered designs and analyses to model intensive longitudinal data (ILD) (Fisher & Boswell, 2016; Wichers, 2014). ISM allows researchers to measure intra-individual variability and may help researchers gain a greater understanding of how intraindividual patterns differ from group level patterns over time (Bringmann et al., 2013; Myin-Germeys et al., 2009). In the past, it was difficult to adequately model intra-individual variability due to limitations in data collection and statistical methodology (as stated in Fisher, 2015). The development of ISM and person-centered analyses have made it possible to design within-subjects studies that can measure individual-specific trends (as discussed in Fisher, 2015). Person-centered analytic methods, such as vector auto-regression (VAR) (e.g., Bringmann et al., 2013) and dynamic structural equation modeling (DSEM) (e.g., Asparouhov, Hamaker, & Muthén, 2018), allow for the analysis of large amounts (> 100 time points) of ILD to construct individual risk models that illustrate person-specific relationships between risk factors over time. In this way, each model represents the results of an individual-specific study (Wichers, 2014). This methodology complements traditional group-based designs and may provide insight into idiographic, individual-specific factors that characterize fluctuations in mood. Furthermore, the pairing of multilevel (i.e., group-level) with idiographic designs allows researchers to accurately model both inter- and intra-individual variability and demonstrate how group-level relationships differ from individual-specific trends. Most importantly, information from individual risk models have the potential to provide personalized and individual-specific directives for psychological care and could be used to improve prevention efforts for anxiety and depression (Fisher, 2015; Wichers, 2014).

Given the dearth of research on idiographic risk factors for depression among individuals with SAD, I aimed to construct idiographic risk models for individuals experiencing comorbid SAD and a current or past major depressive episode. Previous research has demonstrated that individuals who are diagnosed with SAD and a depressive disorder are at increased risk for future depression and are more likely to experience negative outcomes, such as more severe and frequent depressive episodes and greater suicidality (Stein et al., 2001). Thus, all individuals in this study were diagnosed with current SAD and either a current major depressive episode or at least one past major depressive episode. The decision to recruit individuals with current SAD and either a current or past major depressive episode allowed me to examine dynamic fluctuations in affect and emotional experience in a group of individuals who were at increased risk for more severe depressive symptomatology in the future. Importantly, studies using related methodologies have shown that there is still significant variation in affect among individuals who are clearly depressed (Peeters, Berkhof, Delespaul, Rottenberg, & Nicolson, 2006).

As sadness is a hallmark symptom of depression, I aimed to examine the individual-specific dynamic relationships between affective and behavioral risk factors for daily sadness. I hypothesized that maladaptive behaviors, such as social avoidance, or affective states, such as anxiety, would predict daily levels of sadness as reflected by directed longitudinal relationships in an individual risk model. However, I hypothesized that the patterns reflected would differ across individuals. Furthermore, I aimed to construct a multilevel model using ILD to examine risk factors for daily sadness on the group-level. I hypothesized that although individuals may share some pathways in common with the group model, no one individual would demonstrate the exact model structure as the group-level model, highlighting the idiographic nature of an individual's symptomatology.

Chapter 2: Methods

2.1 Participants

Women (N = 35) were recruited from the general university community. Women were recruited as they are more likely to experience internalizing disorders, including SAD and depression (Leach, Christensen, Mackinnon, Windsor, & Butterworth, 2008; Nolen-Hoeksema & Girgus, 1994). Additionally, I would not be able to study the effects of sex or gender due to the limited sample size and thus, only individuals who self-identified as cis-female were recruited. This sample included women (n = 26) who were enrolled in undergraduate courses, including introductory psychology courses, as well as those who were graduate students or employees (n = 9). All women completed a diagnostic clinical interview as part of their participation in a separate study. This separate study aimed to evaluate changes in depression and major depressive episode status over a two-month period of time in a sample selected for higher trait levels of social anxiety. The results of this study are outside the aims of this dissertation and will not be presented or discussed here. Those who met criteria for current SAD and a current (n = 6) or past major depressive episode (n = 29) were invited and participated in this dissertation study. Ages ranged from 18 – 37 years (M = 21.37 years, SD = 5.20), and most women identified as White (51.43%), although 28.57% identified as East Asian and 17.14% identified as Black. Furthermore, 5.71% identified as Hispanic.

2.2 Measures

2.2.1 MINI Interview – 5. The MINI-5 is a structured interview that assesses clinical disorders. Diagnoses correspond to the psychiatric disorders listed in the Diagnostic and Statistical Manual, 5th edition (American Psychiatric Association, 2013) and the International Classification of Diseases, 10th edition (World Health Organization, 1992). The MINI-5 was

used in the separate study to assess clinical disorders. In this separate study, graduate student clinicians assessed participants at two time points, approximately 2.5 months apart. A licensed clinical psychologist (TLR; chair of this dissertation) supervised the graduate student clinicians over the course of this separate study. Inter-rater reliability was assessed using blinded independent raters and by calculating the kappa statistic. Inter-rater reliability was good for SAD ($\kappa = .73$) and excellent for a MDE ($\kappa = .83$).

2.2.2 ISM Items. Daily ISM assessments consisted of 14 items that reflected constructs implicated in the Cumulative Interpersonal Risk Model (Epkins & Heckler, 2011) and in the diagnostic criteria for an MDD, such as sadness, anxiety, irritability, and restlessness. Additionally, physiological or behavioral indicators, such as hunger, physical activity, and social avoidance that may play a role in predicting affect were also included (for a full list of all ISM items, see Appendix A). Participants rated all questions based on their present-moment experience, which was in keeping with previous methodology (e.g., Hektner, Schmidt, & Csikszentmihalyi, 2007; Shiffman, Stone, & Hufford, 2008). Items were administered using a 0 - 10 integer sliding scale (Hektner et al., 2007).[1] Due to concerns for participant burden, items were intentionally limited in number. It was estimated that the entire survey took less than three minutes to complete at each assessment, which is consistent with methods commonly used in related research (e.g., Hektner et al., 2007).

2.3 Procedure

Eligible participants provided informed consent before enrolling in this dissertation study and the Washington University in St. Louis Institution Review Board approved all dissertation study procedures. Participants underwent a brief training protocol administered by the student

[1]There was an error in the scale creation of two ISM items that affected two participants in the sample. These participants used a 0 - 100 integer sliding scale instead of a 0 - 10 integer sliding scale. Responses from these two items were collapsed to form a 0 - 10 integer scale (i.e., a 70 was converted to a 7).

investigator before beginning the study. The initial training session lasted approximately 30 minutes. During this training session, the student investigator guided the participant in setting up the start and end dates for the study, as well as selecting 12-hour period to receive the five daily surveys. Training also included instructions on how to download and use the free smartphone app through which a majority of participants completed surveys.[2] After completing the training, all study protocol took place outside the lab on the participant's smartphone or computer device. ISM assessments were administered over the participant-selected 12-hour time range for 30 days. That is, the same ISM survey was administered five times a day (i.e., every three hours) for approximately a month (total of 150 assessments). During the study, the student investigator sent each participant weekly progress emails, which documented the number of surveys completed and the amount of compensation earned for the week.

At the end of the 30-day period, participants received a final email stating their progress in the study. Participants were instructed to return to the lab to complete the second session of the separate study. At the end of this session, participants were debriefed on the purpose of both studies. Participants received a preliminary version of their personalized model using output generated from graphical VAR (GVAR; Epskamp, 2018) and the student investigator explained any significant model effects. At the end of the debriefing, the participants were thanked for their efforts and completed forms for payment. Participants received monetary compensation in proportion to the number of EMA surveys they completed each day. Maximum compensation was $82.50, which reflected 100% completion of EMA surveys. A majority of participants completed over 100 surveys (M = 125.43, SD = 19.26).

[2]Two smartphone applications were used to administer ISM surveys during the course of this dissertation. The first app used was only compatible with Apple iPhones. During this initial period that this app was used, two participants were recruited who were Android users. To accommodate use of their Android device, these participants completed ISM surveys via a Qualtrics survey link. The remainder of participants completed ISM surveys via a smartphone application (LifeData) that was compatible with both Apple iPhone and Android users.

2.4 Data Analytic Plan

Prior to all study analyses, each participant's data was visually inspected and cleaned. Extraneous variables collected by the apps (e.g., timestamps) were removed and missing data was specified. Additionally, to prepare data for dynamic structure equation modeling (DSEM), three rows of missing data were added between each day to account for the three assessments that would have been administered outside of the 12-hour time range. The addition of these missing rows ensured that the model did not predict trends over overnight periods, which would violate the statistical assumptions of DSEM.

To construct the individual risk model, two analytic methods were used. The first method used was GVAR, accessed through the GVAR package in R (Epskamp, 2018). GVAR has been used in previous idiographic studies (cf. Epskamp et al., 2018) and often relies on a network theory framework[3] to model both contemporaneous and directed relationships between individual items for each participant. The contemporaneous model represents partial correlations between items across time; whereas, the directed model displays partial regressive relationships across time (Epskamp et al., 2018). GVAR also utilizes the least absolute shrinkage and selection operator (LASSO) to select and shrink relationships that are small enough to be considered spurious to zero to highlight the most relevant relationships within the model (Tibshirani, 1996). To account for collinearity between items that may occlude inter-item relationships and to pull together items that may share a common latent factor, individual items were composited if they were correlated at $r > .65$, reflecting a moderately strong relationship. Additionally, if there were correlations ($r > .30$) between time variables and ISM items, the ISM items were regressed onto

[3] Network theory relies on the assumption that individual items direct change amongst themselves, as opposed to an underlying latent variable directing change among individual indicators. Further explanation of network theory and the strengths and limitations of this approach can be found elsewhere (Fried & Cramer, 2017).

time variables and the residuals were entered into the GVAR model in an effort to account for any systematic effects of time. All GVAR analyses were conducted in R using the dplyr, version 0.8.0.1 (Wickham, François, Henry, & Müller, 2019), graphicalVAR, version 0.2-2 (Epskamp, 2018), and ggplot2, version 3.1.0 (Wickham, 2016) packages. Code for GVAR analyses is included in Appendix B.

The second analytic method used was DSEM (Asparouhov et al., 2018; Muthén & Muthén, 1998-2017). DSEM uses a structural equation modeling approach with Bayesian estimation to model direct inter-item relationships, as well as latent variables. Importantly, DSEM is able to directly model effects of time by adding these pathways to the structural equation model. DSEM models included autoregressive and cross-lagged pathways between ISM items. An autoregressive pathway represents the relationship between one variable at two different time points (i.e., x_{-1} predicting x); whereas a cross-lagged pathway represents the relationship between two variables from one time point to the next (i.e., x_{-1} predicting y). Mplus notifies the user of the number of iterations in a DSEM analysis that may contain some violations to stationarity, which assists the user in determining whether the individual's data meets the assumptions time series analysis.

Before beginning DSEM analyses, I planned to use exploratory factor analyses (EFA) using each participant's data to determine whether latent variables should be modeled using DSEM. The EFAs used in this procedure are equivalent to P-technique described in Lee and Little (2012) and up to five factors were specified in each EFA. Each model was examined and output from parallel analyses and cutoffs for standard fit indices were used to determine whether a simple factor structure existed. A simple factor structure refers to a model structure in which there are a sufficient number of indicators loading onto each factor with low cross-loadings on

the other factors (as described in Yang, 2005). If a simple factor structure was identified, I planned to model this factor structure using DSEM. Notably, there were no clear factor structures for participants.

I also planned to construct DSEM models that included all autoregressive and cross-lagged paths between ISM items and between ISM items and time variables (i.e., day number and survey number). However, as the individual DSEM model included over 200 pathways (i.e., more pathways than observations), a simpler model was constructed that included the effects of ISM items on time, as well as a regression path between all ISM items predicting feelings of sadness. This model was chosen because sadness was the primary ISM item of interest.

To construct individual DSEM models, an open model was run without specifying iterations. After the model converged, successive models were run with twice as many iterations and the proportional scale reduction (PSR) factor was evaluated (Muthén, 2010). The model was considered to be final when at least two models converged with low, stable PSR (a cutoff of 1.01 was used). When necessary, commands to prune iterations (Asparouhov, 2014) and weak priors (Linda Muthén, personal communication to Thomas Rodebaugh, 12/13/2017) were used to achieve model convergence. Additionally, variables in which an individual used fewer than 7 response options were specified as categorical. Categorical variables with response options that were only endorsed once were recoded into the nearest response option. This means that if a participant only used the response option 6 once, but used the response option 5 ten times, then the one instance of 6 was recoded into a 5 to assist with model convergence.

To model group-level relationships, multilevel DSEM (ML-DSEM) was used (Hamaker et al., 2018). Results from the ML-DSEM model demonstrated the autoregressive and cross-lagged relationships that are significant at the group-level. I first specified two initial models that

included all random within-level effects, as well as all random effects excluding random covariances between the error and variances of the covariance. These models were too complex to result in convergence (due to the large number of pathways) and thus, a simpler model was run. This simpler model included the random within-level auto-regressive and cross-lagged effects between all ISM items predicting feelings of sadness, as well as regressing all ISM items on time variables. Random error terms were also estimated. On the between-level, the means and variance of each parameter from the within level were included. ML-DSEM also generates individual-level estimates for each pathway using information obtained in the generation of the group-level model. These estimates were used in comparison with the individual-level estimates generated from each idiographic DSEM (see below for further discussion). Mplus, version 8.2 (Muthén & Muthén, 1998-2017) was used for P-technique and DSEM analyses and code for DSEM analyses is included in Appendix B.

To compare overlap between idiographic and multilevel models, I compared the number of pathways that were demonstrated in the multilevel model that were represented in the individual-level models and calculated the percentage of individual-level pathways that were shared with the multilevel model. To assist with data visualization, I constructed density plots of the standardized individual-level estimates computed by the multilevel model compared with the standardized estimates produced in each individual-level model for each pathway predicting down. These graphs served to visualize whether the individual-level estimates from multilevel DSEM versus idiographic DSEM models reflected similar distributions. Plots were generated using R, including the Mplus Automation package, version 0.7-3 (Hallquist & Wiley, 2018) and the ggplot2 package, version 3.1.0 (Wickham, 2016). Code for generating these graphs is included in Appendix B.

2.5 Power Analyses

There were no previous simulation studies to suggest the sample size needed in order to provide sufficient power for these proposed analyses. However, the study design allowed for the collection of over 100 ISM time points, which was in keeping with previous studies of this nature that treat each individual as a single study for individual-level analysis (Wichers, 2014).

Chapter 3: Results

3.1 GVAR Models

Before constructing GVAR models, each individual's data was examined for effects of time. If there was a correlation ($r > .30$) with either day or survey, that time variable was regressed onto all variables and the residuals were entered into the GVAR model. This accounted for systematic effects of time and reduced concerns regarding violations to stationarity. For participants who had effects of day and survey, all variables were regressed onto the time variable that exerted the largest effect (e.g., day). Overall, a majority of individuals ($n = 25$, 71.43%) had effects of day, although a considerable number ($n = 14$, 40.00%) had effects of survey, and fewer ($n = 10$, 28.57%) had effects of both day and survey. Additionally, a majority of individuals ($n = 26$ individuals, 74.29%, $M = 1.06$ variables, $SD = 0.84$, range 0 – 3 variables) had large ($r > .65$) inter-item correlations. These items were composited.

Notably, there were some participants ($n = 11$) for whom multiple variables were correlated ($r > .65$) with one variable, but not all items within this subset were correlated $r > .65$. In these instances, if all items within the subset were correlated $r \geq .60$, all items were composited to create whole-item composites. There was another instance in which several variables were all correlated $r > .65$. In these cases, only items with the largest correlations were composited to prevent a majority of items from being composited into one item. Results will be presented from the non-composited and composited models below. Directed effects from GVAR models are displayed in Figure 1.

Overall, when examining results from the non-composited models, the autoregressive paths were more common than cross-lagged paths. Autoregressive pathways were more frequent

(*n* = 13 individuals, 37.14%; *M* = 0.54 pathways, *SD* = 0.82, range 0 – 3 pathways) compared to cross-lagged pathways (*n* = 9 individuals, 25.71%; *M* = 0.37 pathways, *SD* = 0.73, range 0 – 3 pathways). Results from the non-composited models suggest that the directed GVAR models were notably sparse, with few autoregressive or cross-lagged pathways when examining relationships over the 3-hour time period. Additionally, among those individuals whose model reflected at least one cross-lagged pathway, there were few consistent predictors. For example, only three (out of nine) individuals had a directed pathway that included feelings of sadness (i.e., down). For one individual (ID = 5) feeling down at one time point predicted feeling less happy later on. In contrast, another individual's (ID = 10) feelings of down were predicted by feeling lonely earlier on; whereas, for the third individual (ID = 11), feeling irritable predicted feeling more down three hours later.

When examining results from the composited models, the general pattern of results varied slightly. That is, compositing variables resulted in models with more autoregressive and cross-lagged effects. In contrast to the non-composited models, cross-lagged pathways were more frequent (*n* = 11 individuals, 31.43%; *M* = 0.91 pathways, *SD* = 2.17, range 0 – 10 pathways) compared to autoregressive pathways (*n* =16 individuals, 45.71%; *M* = 0.80 pathways, *SD* = 1.18, range 0 – 5 pathways). Although the use of composite variables hindered comparison of model structure across participants, there appeared to be few shared pathways. This was consistent with results from the non-composited models. As seen in Figure 1, the number of directed pathways differed across individuals.

3.2 DSEM Models

3.2.1 Individual-level factor structure (P-technique). P-technique or EFA was conducted using each participant's data to determine individual-level factor structure. Parallel analyses and

cutoffs for fit indices were examined for the first ten participants to assist in identifying the correct number of factors. In determining the number of factors to use, I attempted to evaluate simple factor structure in which variables loaded clearly onto one factor, with low loadings on other factors, and minimal cross-loadings (as described in Yang, 2005). Overall, there was no simple factor structure for these participants. For a majority of these participants, parallel analyses suggested a number of factors that was not supported by fit indices or vice versa. For participants in which both parallel analyses and fit indices supported the same number of factors, there were typically cross-loadings between these factors or fewer than three indicators that were predicted by a single factor. This impeded my ability to identify a simple factor structure. Although the 14 items reflected a group of related symptomatology and constructs, there was no *a priori* hypothesized factor structure to guide the determination of simple factor structure. Thus, results from this set of analyses were not used in the creation of DSEM models. Only DSEM models containing autoregressive and cross-lagged paths for each variable predicting down were constructed. Pathways between day, survey, and ISM variables were also included.

3.2.2 Lag 1 multilevel and individual-level DSEM models. The Lag 1 multilevel and all DSEM models converged with stable, low PSR across successive models with increasing iterations. Effects from the Lag 1 multilevel model can be seen in Figure 2. Overall, there were effects of time, such that feeling down increased over the month. Additionally, there was an autoregressive effect of feeling down, such that feeling down at one time point predicted feeling more down three hours later. Feeling lonely, avoiding social situations, and amounts of physical activity since the last assessment were all risk factors for feeling more down three hours later. In contrast, feeling calm was a protective factor for feeling down. Although these were the directed pathways demonstrated in the multilevel model, it should be noted that there may be indirect

pathways affecting feelings of sadness that were not modeled, such as one variable affecting loneliness, which in turn predicted feeling more down later on.

Examining each woman's individual-level model revealed considerable variability. Only a minority of individuals (n = 14, 56.00%) had models that demonstrated at least one statistically significant autoregressive or cross-lagged effect (see Figure 3). Of these fourteen individuals, two individuals (IDs 14 and 20) demonstrated models that reflected one directed effect, such that for these two individuals, their feelings of sadness increased over the month. Likewise, there were two individuals (IDs 13 and 22) whose models reflected one significant effect – an autoregressive effect of down – suggesting that for these two individuals, their feelings of sadness were predicted by higher levels of sadness at the previous time point.

The remaining ten women exhibited varying pathways (significant pathways demonstrated in Figure 3). Results from each of these women's models are presented below. When the first woman (ID = 001) felt focused, she was less likely to feel down later on. For the second woman (ID = 004), when she felt anxious at one time point, she was less likely to feel down later on. When the third woman (ID = 006), felt accomplished at one time point, she felt less down later on. Additionally, her sadness increased systematically over the course of the month. For the fourth woman (ID = 007), when she felt hungry, she was less likely to feel down later on. The fifth woman's (ID = 008) Lag 1 model suggested her feelings of loneliness predicted feeling more down later on. The sixth woman's (ID = 011) Lag 1 model suggested that feeling irritated and avoiding social situations led to her feeling more down later on. Her model also suggested that when she felt accomplished, she felt less down later on. When the seventh woman (ID = 023), felt focused, she was more likely to feel down later on. However, her model also contained a negative directed relationship between lonely and down, such that when she felt lonelier, she

felt less down later on. Additionally, she demonstrated a systematic increase of sadness across the month. For the eighth woman (ID = 024), when she avoided social situations she felt less down later on. Additionally, her model demonstrated a negative autoregressive effect, such that feeling down at one time point predicted feeling less down later on. When the ninth woman (ID = 026) felt irritable, she was more likely to feel down later on and when she engaged in greater amounts of physical activity since the last time point, she was also more likely to feel down. Notably, her model also demonstrated a positive autoregressive effect for sadness and a systematic time effect for survey administration. That is, her feelings of sadness predicted feeling more down later on, although her levels of sadness decreased as the day went on. Finally, the tenth woman's (ID = 031) Lag 1 model reflected a negative directed effect between feeling hungry and down later on. Similarly, when she felt calm, she was less likely to feel down at a later time point.

The amount of individual-level pathways that were shared with the group-level model varied and ranged from 0% (i.e., no congruent pathways represented in the group-level model for that individual) to 33% overlap with the group level model. Of the six significant pathways indicated in the group-level model, individuals had few pathways in common with the multilevel model, (M = 0.37, SD = 0.65, range = 0 - 2). The most frequent congruent pathway was the significant effect of down increasing across the month (n = 4, 11.43%), followed by the significant positive autoregressive effect of feeling down (n = 3, 8.57%). Furthermore, there were three instances of pathways that were incongruent in valence with the group level model (e.g., the group-level model had a positive estimate; whereas, the individual-level model contained a negative estimate). One woman demonstrated a model in which her sadness decreased (rather than increased) over the course of the month and social avoidance led to

decreased (rather than increased) sadness later on. For a second woman, feeling lonely predicted less sadness later on. The amount of overlap in multilevel versus individual-level estimates is demonstrated in Figure 4. The estimates from the down autoregressive pathway appear to reflect two distributions with different means. However, the estimates from all other cross-lagged pathways appear to represent a similar distribution with individual-level estimates reflecting a larger spread of distribution compared to the multilevel estimates.

3.2.3. Lag 2 multilevel and individual-level DSEM models. Lag 2 multilevel and idiographic DSEM models were also constructed. The Lag 2 multilevel and all DSEM models converged with stable, low PSR across successive models with increasing iterations. Effects from the Lag 2 multilevel model can be seen in Figure 5. Overall, there was an autoregressive effect of feeling down at Lag 1 and 2, such that feeling down at one time point predicted feeling more down both at three and six hours later. Consistent with the Lag 1 multilevel model, feeling lonely predicted feeling more down three hours later, whereas, feeling calm predicted feeling less down later on. Again, as with the Lag 1 model, there may be indirect pathways affecting feelings of down that were not modeled.

Similar to the Lag 1 models, there was considerable variability to the number of significant directed pathways demonstrated at Lag 2 for each woman. There were nine (25.71%) women whose model demonstrated at least one significant directed Lag 2 pathway and three of these women demonstrated a significant Lag 2 autoregressive effect. Interestingly, two out of the three women's autoregressive pathways were incongruent in valence from the multilevel model. That is, for these two women when they were feeling down, they were less likely to feel down six hours later.

Examining the Lag 2 pathways in each woman's model revealed different patterns

(significant pathways demonstrated in Figure 6). There were eight women whose models included a significant Lag 2 pathway between an ISM variable and feeling down (e.g., cross-lag) and the results from these models are presented here. For example, in the first woman's (ID = 009) Lag 2 model, there was a negative relationship between feeling happy at one time point and feeling down six hours later. A second woman's (ID = 011) Lag 2 model demonstrated that when she felt drowsier, she felt more down six hours later. Interestingly, when she felt more focused, she also felt more down six hours later. A third woman's (ID = 021) Lag 2 model reflected that when she felt calm at one time point, she felt less down six hours later. Her model also demonstrated a positive autoregressive relationship, such that feeling down at one time point predicted feeling more down later on. A fourth woman's (ID = 024) Lag 2 model included pathways suggesting that feeling calm predicted feeling less down two time points later. However, feeling anxious also predicted feeling less down two time points later. Additionally, for this woman feeling focused, lonely, and avoiding social situations predicted feeling more down six hours later. Furthermore, her model reflected a negative autoregressive effect, such that feeling down at one time point predicted feeling less down six hours later. Additionally, the fifth woman's (ID = 025) Lag 2 model demonstrated two pathways predicting down. One pathway suggested that feeling happy at one time point predicted feeling less down two time points later. However, a second pathway suggested that feeling pleased predicted feeling more down six hours later. The sixth woman's (ID = 028) Lag 2 model suggested that her irritability predicted feeling more down two time points later. A seventh woman's (ID = 033) Lag 2 model reflected pathways in which feeling anxious and avoiding social situations predicted feeling down later on, yet feeling accomplished also predicted feeling down two time points later. Finally, the eighth woman's (ID = 035) Lag 2 model suggested that when she avoided social situations, she felt less

down six hours later.

3.2.4 DSEM Post-hoc tests. To address issues of multicollinearity within the DSEM models, correlation pathways from the multilevel and individual-level DSEM models were examined and correlation pathways that were $r > .80$ were noted. There were no correlation pathways $r > .80$ in the multilevel DSEM models. There were six (17.14%) women whose idiographic DSEM models contained correlation pathways that were $r > .80$. Examining the correlations in these women's models revealed that feeling happy and pleased with one's experience were correlated $r > .80$ for each of these women. For two of these women, other items in addition to pleased and happy were also correlated $r > .80$. I created a latent factor for all items that were correlated $r > .80$. Latent factor and non-latent factor DSEM models revealed identical results for four of the six women, suggesting that the model structure did not differ substantially after accounting for the high correlations between select items. However, the latent factor DSEM models did reveal slightly different results for two women. For one woman, her latent factor DSEM revealed an autoregressive effect of sadness instead of an effect of day on feeling of sadness. For the other woman, her latent-factor DSEM model revealed a positive, directed relationships between focused and down, instead of a negative directed relationship between calm and down.

Chapter 4: Discussion

In this dissertation, I collected ILD from a group of women with SAD and a major depressive episode and constructed individual-level and multilevel risk models of daily sadness using DSEM. I also constructed individual risk networks using GVAR for each woman. I hypothesized that maladaptive behaviors, such as social avoidance, or affective states, such as

anxiety, would predict daily levels of sadness as reflected by directed longitudinal relationships in an individual risk model, but that the number and pattern of directed effects would reflect an idiographic constellation of risk factors. Furthermore, I hypothesized that these patterns would differ both between individuals and from the group-level model. Results largely supported my initial hypotheses and revealed clear directions for improving the use of idiographic methodology. Findings from individual risk models constructed using GVAR and DSEM demonstrated systematic effects of time. Additionally, results from both methods highlighted intraindividual variability in that no two individuals demonstrated the exact same model. Furthermore, there was minimal overlap between multilevel and individual-level DSEM pathways. Likewise, although the individual-level estimates estimated from multilevel and individual-level DSEM models largely reflected similar distributions, the spread of the individual-level DSEM estimates was generally much larger than the spread of the individual-level ML-DSEM estimates. Overall, results suggest clear evidence of idiographic variability in mood and emotional experience among women with SAD and a history of a major depressive episode.

Examining the Lag 1 multilevel model revealed an effect of time, as feelings of sadness increased systematically over the course of the month on average. Additionally, there was also an autoregressive effect of down, suggesting that on average, when a woman felt sad at one time point, she was more likely to feel sad at the following time point. Likewise, women who were less calm and lonelier were more likely to feel down three hours later. Furthermore, there were lagged relationships between sadness and two behavioral indicators of mood – the amount of physical activity since the last time point, and amount of social avoidance. That is, greater physical activity since the last time point in addition to greater social avoidance predicted

increased feelings of sadness later on. When examining the Lag 2 multilevel model, the Lag 1 effects for (less) calm and lonely remained. Additionally, there was a Lag 1 and Lag 2 autoregressive effect. Overall, the only Lag 2 specific pathway was the effect of sadness predicting itself six hours later.

Some of these multilevel results are in keeping with the social and interpersonal factors implicated in the Cumulative Interpersonal Risk Model. In their paper describing this theory, Epkins and Heckler (2011) cited studies that support the unique associations of loneliness to both social anxiety and depression for adolescent women and highlighted the role of loneliness in maintaining depression. Thus, it is fitting that feelings of loneliness predicted feeling down three hours later in both the Lag 1 and Lag 2 models. However, these findings may be inconsistent with results demonstrating the efficacy of physical activity on mood and theories of behavioral activation, in which individuals are encouraged to engage in activities that are associated with positive affect and accomplishment. For example, there were no significant directed multilevel pathways and few idiographic pathways between feeling pleased with one's experience, feeling happy, physical activity, or feelings of accomplishment and sadness over the course of three hours (Lag 1) or six hours (Lag 2). Furthermore, in the Lag 1 multilevel model, amounts of physical activity since the last time point positively predicted feelings of sadness, which is in contrast to previous studies suggesting that physical activity may be beneficial for mood (Peluso & Guerra De Andrade, 2005).

It is unclear why engagement in physical activity would predict increased feelings of sadness. This finding may be due to how physical activity was defined in this study versus previous studies. For example, in this dissertation, participants were encouraged to report on any type of physical activity engaged in since the last time point. Specific activities like walking,

exercise, or stretching were noted in the description of this ISM item; however, participants were encouraged to consider and report on physical activity more broadly (e.g., household chores, walking to class, going to the gym, or going out dancing). Previous literature describing the acute and beneficial effects of physical activity on mood have largely defined physical activity as aerobic exercise or running (Yeung, 1996) and, thus, results from those studies may not generalize to this dissertation, as participants may have reported on lower intensity aerobic exercise or physical activity more generally. Furthermore, the activities included in the definition of physical activity described above each carry different positive or negative reinforcements. For example, an individual may find that going out dancing is more enjoyable and positively reinforcing than walking to class. The behavioral activation literature refers to the beneficial effect of physical activity to the extent that it is positively reinforcing and in line with the patient's values (Lejuez & Hopko, 2013). Notably, other researchers have demonstrated a differential impact of physical activity on positive affect rather than negative affect (Mata et al., 2012; Pasco et al., 2011).Thus, it may not be surprising that physical activity positively predicted feeling sad later on if participants were reporting on physical activity that was not positively reinforcing.

Examining individual level models using GVAR reflected a relative sparseness of directed pathways, which was largely consistent with the results when constructing individual-level DSEM. For example, there were few directed predictors of sadness in the GVAR models. Likewise, across the idiographic DSEM models, fewer than half of the participants exhibited a single directed pathway. There are several hypotheses that may help to account for the sparseness of the models. In regards to the sparsity of directed pathways in the DSEM models, it is important to note that only direct predictors of sadness were modeled. That is, there could be

indirect predictors of sadness (e.g., irritability predicting physical activity, which predicts sadness). I chose not to model indirect predictors in an effort to increase model parsimony and to reduce concerns regarding Type I error. Future studies using a larger number of observations are necessary to adequately model all indirect effects and inter-item relationships in order to better understand predictors of sadness.

Although indirect pathways were not modeled in the DSEM approach, all inter-item pathways were modeled using GVAR. Results from the GVAR models were largely similar to the results seen in DSEM, in that few women exhibited directed pathways. This pattern of results could be due to the use of the LASSO approach, which uses the *bet on sparsity* principle (Hastie, Tibshirani, & Friedman, 2001) to shrink pathways with small effect sizes to zero. Accordingly, some amount of model sparsity should be expected and models produced using the LASSO approach often have low sensitivity, but high specificity (Epskamp, Kruis, & Marsman, 2017). That is, I can be reasonably sure that the directed pathways revealed in the GVAR models here are true pathways demonstrated in the model, but I cannot be sure that this is the only potential model structure. However, it is important to note that the GVAR results seen here are sparser than would be expected from using the LASSO approach alone, and there are likely other factors discussed below that additionally account for model sparseness.

Another potential explanation for the sparseness may concern the way time was accounted for in the GVAR models. In these models, a time variable was regressed onto all ISM items if any ISM item and that time variable were correlated $r > .30$. In these instances, the residuals were entered into the GVAR model. Arguably this is a crude method for modeling time, as compared to directly modeling the effects of time as was done for the individual-level DSEM models. For example, in a given individual, not all ISM items were correlated with a time

variable at $r > .30$. Additionally, only the time variable with the largest effect on a given ISM item was modeled. Thus, if an individual's ISM data was correlated $r > .30$ with both day and survey, only the effects of the time variable with the largest effect were accounted for. However, although time was not modeled directly in the GVAR models, the sparse results are still largely consistent with the DSEM models, in which time was modeled directly.

A third reason for the relative sparseness in the idiographic models could be due to potential effects of multicollinearity. For example, if ISM items were highly correlated, this may have suppressed directed effects. This concern was addressed through the use of composites in the GVAR models, which will be discussed later on. Although no composites were used in the DSEM models, the correlational pathways were examined to determine if there were any ISM items that were correlated $r > .80$ (suggesting concerns with multicollinearity). There were six women whose individual-level DSEM models revealed high correlations between pleased and happy. This suggests that these specific women may have interpreted these two ISM items similarly. However, results from the individual-level DSEM models did not differ substantially after these high correlations were accounted for.

Although there was minimal change in DSEM models after accounting for high correlations, there did appear to be an effect of compositing within the GVAR models. Examining the composited GVAR models often revealed more directed pathways compared to the non-composited GVAR models. It could be that the use of composites increased statistical power to model directed pathways. Likewise, is possible that the indicators composited in the GVAR models were actually predicted by a shared latent variable that was not addressed in the non-composited models. That is, although GVAR cannot model latent variables directly, the use of composites may have achieved a similar effect. This is supported by the fact that the items that

were composited in GVAR nearly always loaded onto the same factor in the P-technique analyses. However, it should be noted that in the P-technique analyses, there were often additional ISM items that tended to load onto the same factor as the composited indicators (i.e., there was not a perfect match between suggested factors from P-technique and the composites).

The minimal overlap between multilevel and individual-level DSEM models was also notable. When comparing the Lag 1 multilevel and individual-level DSEMs, systematic increases in feeling down over the course of the month was the most frequent pathway across individual Lag 1 DSEM models, followed by a positive autoregressive effect. These effects were consistent with the Lag 1 multilevel model. However, it should be noted that these pathways were only significant in fewer than 20% of the sample. Importantly, no one individual shared all significant pathways with the group-level model and the percent overlap with the Lag 1 multilevel model ranged from 0% to 33%. Notably, when significant pathways were represented in both an individual and in the multilevel model, the pathway was more likely to be congruent (i.e., of similar valence) than incongruent (i.e., of different valence). However, it was more likely for an individual level effect to not be represented in the multilevel model than to be represented and vice versa.

These observations suggest that there is minimal overlap between the multilevel and individual level models, which supports the hypothesis that individuals would differ from each other and from the group. However, because there have been few studies that have examined multilevel and individual-level DSEM models in tandem, these results necessitate careful consideration to determine which level of model (i.e., multilevel versus individual-level) provides the most useful information regarding individuals. It could be that the multilevel model was not capable of accurately modeling individuals within the group due to erroneous

assumptions about individuals. Alternatively, it could be that the individual-level models were inaccurate due to low statistical power and higher variance. Thus, measures of central tendency and dispersion were examined using individual-level estimates from both the individual-level and multilevel models.

Individual-level estimates for each pathway predicting sadness were generated in both the multilevel and individual-level DSEM models. Comparing the individual-level estimates generated from the multilevel model with the idiographic estimates generated from the individual-level DSEM models could help to determine the nature of the differences between the multilevel and individual-level DSEM models. For example, if there were true differences in the group versus individual-level models these differences could manifest as two distinct distributions of individual-level estimates with different means and narrow spreads. Alternatively, if the discrepancy resulted from higher variance within the idiographic models, then there could be one unified distribution of individual-level estimates with one mean, in which the spread of the distribution of the idiographic estimates was larger than the spread of the distribution of the individual-level estimates generated from the multilevel model.

Examining the spread of the distribution for the cross-lagged pathways suggested that the discrepancy between multilevel and idiographic estimates may have resulted from higher variance within the idiographic models (Figure 4). That is, with one exception, the distribution of estimates took a similar shape; however, the spread of the distribution from the idiographic pathways was much larger than the spread of the distribution from the individual-level estimates generated from the multilevel models. This pattern suggests that the individual-level estimates generated from the multilevel model exhibited generally less variance than the individual-level estimates, which is consistent with the fact that the multilevel model had more statistical power

than the individual-level models. Interestingly, the difference in magnitude of the estimates did not appear to differ substantially based on the participant's number of completed assessments, although the magnitude of the estimates from one participant who completed fewer than 60 assessments did appear to differ (see Figure 4) and this discrepancy may have either been due to limited power in the idiographic DSEM model or because individuals with fewer time points are pulled towards the mean in a multilevel model. Additionally, when examining the autoregressive path, there appeared to be two distinct distributions with similar spreads. This finding suggested that the multilevel model may predict autoregressive paths for sadness differently than the idiographic DSEMs, and without additional research discussed below, I am unable to determine which model represents the most accurate or *true* autoregressive paths.

Examining the spread of individual-level estimates revealed that, for the most part, the idiographic estimates were much more variable, which is in keeping with previous research by Fisher and colleagues (2018) who demonstrated that variance around a mean estimate was much larger within individuals than within groups. They argue that this difference is the result of true individual-level variance, reflecting the nature of idiography (Fisher et al., 2018). Similar results were seen in another previous paper, in which individual-level interquartile range was large, again, highlighting the heterogeneity among individuals (Zimmerman et al., in press). Thus, the large spread of the idiographic estimates seen here is to be expected and is in keeping with previous literature. However, the comparison between the individual-level estimates from the multilevel versus idiographic DSEMs advances upon observations from previous research. Previous studies have not compared the spread from individual estimates generated from idiographic and multilevel models. Overall, the spread of individual-level estimates generated from the multilevel model exhibited a similar pattern with a more narrow spread. These results

are fitting, given that the multilevel model had more statistical power than the idiographic DSEMs. However, I cannot be sure whether the higher variance demonstrated in the individual-level DSEM models is due to higher error variance or higher true variance and, therefore, I cannot determine whether the multilevel model is any more accurate than the individual-level DSEM model. Implications of this finding will be discussed later on.

It is notable that the distribution of individual-level estimates for the down autoregressive parameter appeared to differ based on the source of the estimates. It is difficult to determine why these estimates differed in their distributions. A previous group of simulation studies conducted by Liu (2017) suggested that multilevel autoregressive models may outperform idiographic autoregressive models when all individuals in the sample exhibited the same autoregressive parameter (e.g., everyone's down autoregressive parameter is best modeled as a Lag 1 parameter, on a 3-hour time scale). Additionally, in the event that there are heterogeneous autoregressive patterns within the sample (e.g., some people's down autoregressive parameter is best modeled as Lag 1, but others' down autoregressive parameter is best modeled as Lag 2), a Lag 2 multilevel model could outperform the idiographic models (Liu, 2017). However, without testing models with higher lags (e.g., Lag 3, Lag 4) and comparing across the lagged models, it is difficult to determine which lagged autoregressive parameter best suits the women within this group. Thus, in the absence of additional simulation data modeling and comparing autoregressive parameters in this sample using ML-DSEM and DSEM, it is an open question as to whether the multilevel or idiographic DSEM models in this study best models the down autoregressive relationship.

This study demonstrated key strengths in that it is one of the first studies to construct longitudinal individual risk models of psychopathology using a clinical sample and to compare

these individual level models with a multilevel model. These results provide evidence of key differences between the group and individuals within the group. Furthermore, this study used the latest time series methodology available for modeling longitudinal intraindividual variability. DSEM methods improve upon previous methodology in that we can identify some violations to stationarity and note when data does not meet the assumptions of the given time series approach. Additionally, GVAR methodology allowed for construction of the full model and the use of LASSO reduced concern for Type 1 error in modeling numerous inter-item relationships.

However, this study is limited in that I was only able to model pathways predicting feelings of sadness in the DSEM model, in an effort to reduce model complexity. Furthermore, I was not able to identify a simple factor structure using P-technique, which would have allowed me to further reduce modeling complexity by constructing DSEMs using latent variables. Additionally, there were some limitations noted in the GVAR approach, specifically issues in accounting for time and large inter-item relationships (i.e., composites) that may limit the GVAR results. Because ILD was collected via self-report, data may be limited by participant insight. For example, although the pleased with experience and happy items were intended to assess separate constructs, these items were highly correlated in six women, suggesting that these specific women responded to these two items as if they were the same item. Furthermore, because these models rely on self-report, they are only as accurate as the items that are included. Thus, there may have been other idiographic items that would have better captured the dynamic relationship between sadness for specific individual. Finally, comparison of the distribution of estimates between idiographic and multilevel models revealed that the idiographic estimates may be particularly susceptible to higher variance, and suggests that more time points may be necessary to increase the statistical power available to determine the presence of idiographic effects in the

idiographic DSEMs, should they exist. Alternatively, the discrepancy in distributions between the idiographic and multilevel estimates may be accounted for by potentially erroneous assumptions that the multilevel model makes about individuals, especially individuals with fewer time points.

This dissertation is one of the first studies to utilize ILD to examine patterns in emotional experience on both the group and individual level. Evaluating the overlap between results on the group and individual level raises important questions about the clinical utility of these findings. For example, given that so few individuals demonstrated similar individual-level relationships as the group (e.g., no individuals demonstrated the same positive cross-lagged relationship between feeling lonely and feeling sad that was seen on the group level), how should clinicians move forward with translating group-level results to the individual level? For example, could a clinician safely assume that his or her client's loneliness predicts depression, as seen in previous studies and on the group level in this study? Furthermore, for one individual, her levels of loneliness predicted lower (rather than higher) depression. This relationship suggests that if individual risk models were to be used in treatment planning, it would be just as important for the clinician to identify nonexistent or unexpected pathways as expected pathways. In this scenario, it would be useful for the clinician to note whether the client's model reflects a significant pathway between loneliness and sadness in the hypothesized direction, if the pathway is reflected at all. One possibility is that the participant for whom there was a negative directed relationship is already using coping strategies to manage feelings of loneliness that are having an ameliorative effect on her sadness. For example, she may feel lonely when spending time alone; however, spending time alone also facilitates mood-regulation activities (e.g., meditation) that then decrease her sadness later on.

The comparison between the estimates generated from the idiographic models and the multilevel model also provides guidance for how clinical scientists can utilize ILD in future research and clinical applications going forward. For example, the finding that the multilevel model generated individual-level estimates that were of a similar pattern with a more narrow spread than the idiographic models suggests that they may be able to more adequately model intraindividual variability due to greater statistical power. Because ML-DSEM requires fewer observations for each individual, the estimation procedure relies on the power of the group, rather than the single individual. Additionally, unpublished data from a previous study suggested that multilevel models that include a greater number of random paths and error terms produce estimates that are more similar to estimates from idiographic models (in which all pathways are freely estimated) (Rodebaugh, Piccirillo, Gerull, Kallogjeri, & Piccirillo, 2019). Going forward, it may be more advantageous to use ML-DSEM to estimate individual-level relationships, rather than estimating idiographic models in isolation. Combining smartphone technology with the open science framework, it may be possible to gather enough ILD to create a multilevel model that can be shared across multiple clinics and institutions. ILD from a single individual could then be modeled using the multilevel framework to provide more individual-level estimates. This information could then inform the use of self-help and therapeutic interventions.

However, before we can begin using multilevel models to describe intraindividual variability, we must first be sure that the multilevel model is accurately describing each individual within the group. For example, one reason that the idiographic estimates produced a distribution with a wider spread than the individual-level estimates from the multilevel model could have been because the idiographic models had lower statistical power. However, it is equally likely that the multilevel model relied on inaccurate assumptions regarding the individual

resulting in the discrepancy in the two distributions. Future research integrating ILD with experimental design could assist in determining whether group-level results accurately describe individuals. For example, if the multilevel model demonstrated that social stress predicted sadness over time, it would be important to ensure that an intervention designed to experimentally induce social stress resulted in sadness for each individual within the group.

Clearly there is much work to be done before we can begin to integrate idiographic methodology into clinical practice. One particularly important area for future research is examining the time course of mood, symptoms, and emotional experiences. These results reflect relationships demonstrated when items are sampled every three hours. It seems likely that if we were to have assessed these items on a different schedule, a different pattern of results may have emerged. It is difficult to say whether or not the individual level relationships would continue to differ from group-level relationships to the same degree. Future work in the field of affective science is necessary to determine the modal time course for specific emotions. Once the modal time course is determined for the emotion (or the emotion within the person), the appropriate time series method could be used. For example, DSEM could be used to model relationships between items that were assessed on different schedules; whereas, as of now, GVAR cannot.

The issues with intensive longitudinal designs and individual sampling methodology discussed here provide clear avenues for future research. Pursuing these issues in future work will provide evidence to answer key questions in the field of idiography – that is, do group-level models of psychopathology ever fully describe individuals within the group, and if so, when and under what circumstances? Future research in individual-level methodology is necessary to inform and update group-level models of psychopathology using ILD. For example, results from simulation studies may be helpful to construct models with enough statistical power to examine

multiple pathways among various items of interest. Additionally, intervention and experimental studies that utilized ILD would be helpful in assessing whether each individual within the group responds similarly to a given intervention. Results from studies relying on an experimental design would help to determine how well multilevel models depict individual-level findings. Information on how well multilevel models predict intraindividual variability could increase our confidence that differences between group and individual-level models are due to true idiographic differences, rather than limitations in statistical power or modeling approaches. Finally, results from future work can continue to inform the extent to which these findings can be used in clinical settings. With advances in statistical methodology, increases in access to smartphone technology, and accessibility across clinical settings and institutions via the open science framework, individual level ILD has the potential to become increasingly accessible. With a better understanding of how to harness individual-level findings, we can work towards improving personalized directives for psychological care in the future.

References

Acarturk, C., de Graaf, R., van Straten, A., ten Have, M., & Cuijpers, P. (2008). Social phobia and number of social fears, and their association with comorbidity, health-related quality of life and help seeking. *Social Psychiatry and Psychiatric Epidemiology*, *43*, 273–279. http://doi.org/10.1007/s00127-008-0309-1

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., … Lapkin, J. B. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*, *109*, 403–418. http://doi.org/10.1037/0021-843X.109.3.403

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders (5th Ed.)* Arlington, VA: American Psychiatric Publishing.

Asparouhov, T. (2014). Specifying burn in for Bayes. Retrieved March 21, 2019, from http://www.statmodel.com/discussion/messages/11/20726.html?1547519147

Asparouhov, T., Hamaker, E. L., & Muthén, B. (2018). Dynamic structural equation models. *Structural Equation Modeling: A Multidisciplinary Journal*, *25*, 359–388. http://doi.org/10.1080/10705511.2017.1406803

Bringmann, L. F., Vissers, N., Wichers, M., Geschwind, N., Kuppens, P., Peeters, F., … Tuerlinckx, F. (2013). A network approach to psychopathology: New insights into clinical longitudinal data. *PLoS ONE*, *8*, 1–13. http://doi.org/10.1371/journal.pone.0060188

Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin*, *140*, 816–845. http://doi.org/10.1037/a0034733

Dalrymple, K. L., & Zimmerman, M. (2011). Age of onset of social anxiety disorder in depressed outpatients. *Journal of Anxiety Disorders*, *25*, 131–137. http://doi.org/10.1016/j.janxdis.2010.08.012

Epkins, C. C., & Heckler, D. R. (2011). Integrating etiological models of social anxiety and depression in youth: Evidence for a Cumulative Interpersonal Risk Model. *Clinical Child and Family Psychology Review*, *14*, 329–376. http://doi.org/10.1007/s10567-011-0101-8

Epskamp, S. (2018). GraphicalVAR: Graphical VAR for experience sampling data. R package version 0.2.2. https://cran.r-project.org/web/packages/graphicalVAR/graphicalVAR.pdf

Epskamp, S., Kruis, J., & Marsman, M. (2017). Estimating psychopathological networks: Be careful what you wish for. *PLoS ONE*, *12*, e0179891. http://doi.org/10.1371/journal.pone.0179891

Epskamp, S., van Borkulo, C. D., van der Veen, D. C., Servaas, M. N., Isvoranu, A.-M., Riese, H., & Cramer, A. O. J. (2018). Personalized network modeling in psychopathology: The importance of contemporaneous and temporal connections. *Clinical Psychological Science*, *6*, 416–427. http://doi.org/10.1177/2167702617744325

Fisher, A. J., & Boswell, J. F. (2016). Enhancing the personalization of psychotherapy with dynamic assessment and modeling. *Assessment*, *23*, 496–506. http://doi.org/10.1177/1073191116638735

Fisher, Aaron J. (2015). Toward a dynamic model of psychological assessment: Implications for personalized care. *Journal of Consulting and Clinical Psychology*, *83*, 825–836. http://doi.org/10.1037/ccp0000026

Fisher, Aaron J., & Bosley, H. G. (2015). Personalized assessment and treatment of depression. *Current Opinion in Psychology*, *4*, 67–74. http://doi.org/10.1016/j.copsyc.2015.03.031

Fisher, Aaron J., Medaglia, J. D., & Jeronimus, B. F. (2018). Lack of group-to-individual generalizability is a threat to human subjects research. *Proceedings of the National Academy of Sciences of the United States of America, 115,* E6106-E6115. http://doi.org/10.1073/pnas.1711978115

Fried, E. I., & Cramer, A. O. J. (2017). Moving forward: Challenges and directions for psychopathological network theory and methodology. *Perspectives on Psychological Science*, *12*, 999–1020. http://doi.org/10.1177/1745691617705892

Greenberg, P. E., Kessler, R. C., Birnbaum, H. G., Leong, S. A., Lowe, S. W., Berglund, P. A., & Corey-Lisle, P. K. (2003). The economic burden of depression in the United States. *The Journal of Clinical Psychiatry*, *64*, 1465–1475. http://doi.org/10.4088/JCP.v64n1211

Hallquist, M. N., & Wiley, J. F. (2018). Mplus Automation: An R package for facilitating large-scale latent variable analyses in Mplus. *Structural Equation Modeling*, *25*, 621–638. http://doi.org/10.1080/10705511.2017.1402334

Hamaker, E.L., Asparouhov, T., Brose, A., Schmiedek, F., & Muthén, B. (2018). At the frontiers of modeling intensive longitudinal data: Dynamic structural equation models for the affective measurements from the COGITO study. *Multivariate Behavioral Research*, 1–22. http://doi.org/10.1080/00273171.2018.1446819

Hamaker, Ellen L. (2012). Why researchers should think "within-person": A paradigmatic rationale. In M. R. Mehl & T. S. Conner (Eds.), *Handbook of Research Methods for Studying Daily Life* (pp. 43–61). New York, NY: Guilford Press.

Hastie, T., Tibshirani, R., & Friedman, J. (2001). Ensemble Learning. In *The Elements of Statistical Learning* (pp. 605–624). New York, NY: Springer.

Hektner, J. M., Schmidt, J. A., & Csikszentmihalyi, M. (2007). *Experience sampling method:*

Measuring the quality of everyday life. Thousand Oaks, CA: SAGE Publications Inc.

Hirshfeld-Becker, D., Micco, J., Henin, A., Bloomfield, A., Biederman, J., & Rosenbaum, J. (2008). Behavioral inhibition. *Depression and Anxiety*, *25*, 357–367. http://doi.org/10.1002/da.20490

Hirshfeld-Becker, D. R. (2010). Familial and temperamental risk factors for social anxiety disorder. *New Directions for Child and Adolescent Development*, *127*, 51–65. http://doi.org/10.1002/cd.262

Jacobs, N., Myin-Germeys, I., Derom, C., Delespaul, P., van Os, J., & Nicolson, N. A. (2007). A momentary assessment study of the relationship between affective and adrenocortical stress responses in daily life. *Biological Psychology*, *74*, 60–66. http://doi.org/10.1016/j.biopsycho.2006.07.002

Kessler, R. C. (1997). Prevalence, correlates, and course of minor depression and major depression in the National Comorbidity Survey, *45*, 19–30. http://doi.org/10.1016/S0165-0327(97)00056-6

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Chatterji, S., Lee, S., Ormel, J., … Wang, P. S. (2011). The global burden of mental disorders: An update from the WHO World Mental Health (WMH) surveys. *Epidemiologia e Psichiatria Sociale*, *18*, 23–33. http://doi.org/10.1017/S1121189X00001421

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 593–602. http://doi.org/10.1001/archpsyc.62.6.593

Kessler, R. C., Merikangas, K. R., & Wang, P. S. (2007). Prevalence, comorbidity, and service

utilization for mood disorders in the United States at the beginning of the twenty-first century. *Annual Review of Clinical Psychology*, *3*, 137–158. http://doi.org/10.1146/annurev.clinpsy.3.022806.091444

Leach, L. S., Christensen, H., Mackinnon, A. J., Windsor, T. D., & Butterworth, P. (2008). Gender differences in depression and anxiety across the adult lifespan: The role of psychosocial mediators. *Social Psychiatry and Psychiatric Epidemiology*, *43*, 983–998. http://doi.org/10.1007/s00127-008-0388-z

Lejuez, C. W., & Hopko, D. R. (2013). *Brief Behavioral Activation (BA) Treatment Manual - Revised*. College Park, Maryland: University of Maryland College Park.

Liu, S. (2017). Person-specific versus multilevel autoregressive models: Accuracy in parameter estimates at the population and individual levels. *British Journal of Mathematical and Statistical Psychology*, *70*, 480–498. http://doi.org/10.1111/bmsp.12096

Mata, J., Thompson, R. J., Jaeggi, S. M., Buschkuehl, M., Jonides, J., & Gotlib, I. H. (2012). Walk on the bright side: Physical activity and affect in major depressive disorder. *Journal of Abnormal Psychology*, *121*, 297–308. http://doi.org/10.1037/a0023533

Molenaar, P. C. M. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement*, *2*, 201–218. http://doi.org/10.1207/s15366359mea0204_1

Muthén, B., & Muthén, L. (2018). Mplus, version 8.2. Los Angeles, CA: Muthén & Muthén.

Muthén, Bengt. (2010). *Bayesian analysis in Mplus: A brief introduction, version 3.0*. Retrieved from https://www.statmodel.com/download/IntroBayesVersion%203.pdf

Myin-Germeys, I., Oorschot, M., Collip, D., Lataster, J., Delespaul, P., & van Os, J. (2009). Experience sampling research in psychopathology: Opening the black box of daily life.

Psychological Medicine, *39*, 1533–1547. http://doi.org/10.1017/S0033291708004947

Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin*, *115*, 424–443. http://doi.org/10.1037/0033-2909.115.3.424

Pasco, J. A., Jacka, F. N., Williams, L. J., Brennan, S. L., Leslie, E., & Berk, M. (2011). Don't worry, be active: Positive affect and habitual physical activity. *Australian and New Zealand Journal of Psychiatry*, *45*, 1047–1052. http://doi.org/10.3109/00048674.2011.621063

Peeters, F., Berkhof, J., Delespaul, P., Rottenberg, J., & Nicolson, N. A. (2006). Diurnal mood variation in major depressive disorder. *Emotion*, *6*, 383–391. http://doi.org/10.1037/1528-3542.6.3.383

Peluso, M. A. M., & Guerra De Andrade, L. H. S. (2005). Physical activity and mental health: The association between exercise and mood. *CLINICS*, *60*, 61–70. http://doi.org/10.1590/S1807-59322005000100012

Pini, S., Cassano, G. B., Simonini, E., Savino, M., Russo, A., & Montgomery, S. A. (1997). Prevalence of anxiety disorders comorbidity in bipolar depression, unipolar depression and dysthymia. *Journal of Affective Disorders*, *42*, 145–153. http://doi.org/10.1016/S0165-0327(96)01405-X

Rapee, R. M., & Spence, S. (2004). The etiology of social phobia: Empirical evidence and an initial model. *Clinical Psychology Review*, *24*, 737–767. http://doi.org/10.1016/j.cpr.2004.06.004

Rodebaugh, T. L., Piccirillo, M. L., Gerull, K. M., Kallogjeri, D., & Piccirillo, J. F. (2019). Individual variation in tinnitus models as a potential predictor of intervention response. Manuscript submitted for publication.

Rosmalen, J. G. M., Wenting, A. M. G., Roest, A. M., de Jonge, P., & Bos, E. H. (2012). Revealing causal heterogeneity using time series analysis of ambulatory assessments. *Psychosomatic Medicine*, *74*, 377–386. http://doi.org/10.1097/PSY.0b013e3182545d47

Rudolph, K. D., Flynn, M., & Abaied, J. L. (2008). A developmental perspective on interpersonal theories of youth depression. In B. L. Hankin & J. R. Z. Abela (Eds.), *Handbook of Depression in Children and Adolescents* (pp. 79–102). New York, NY: Guilford Press.

Shahar, B., & Herr, N. R. (2011). Depressive symptoms predict inflexibly high levels of experiential avoidance in response to daily negative affect: A daily diary study. *Behaviour Research and Therapy*, *49*, 676–681. http://doi.org/10.1016/j.brat.2011.07.006

Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological momentary assessment. *Annual Review of Clinical Psychology*, *4*, 1–32. http://doi.org/10.1146/annurev.clinpsy.3.022806.091415

Starr, L. R., & Davila, J. (2008). Differentiating interpersonal correlates of depressive symptoms and social anxiety in adolescence: Implications for models of comorbidity. *Journal of Clinical Child & Adolescent Psychology*, *37*, 337–349. http://doi.org/10.1080/15374410801955854

Starr, L. R., Hammen, C., Connolly, N. P., & Brennan, P. A. (2014). Does relational dysfunction mediate the association between anxiety disorders and later depression? Testing an interpersonal model of comorbidity. *Depression and Anxiety*, *31*, 77–86. http://doi.org/10.1002/da.22172

Stein, M. B., Fuetsch, M., Müller, N., Höfler, M., Lieb, R., & Wittchen, H.-U. (2001). Social anxiety disorder and the risk of depression: A prospective community study of adolescents

and young adults. *Archives of General Psychiatry*, *58*, 251–256. http://doi.org/10.1001/archpsyc.58.3.251

Stewart, R. E., & Chambless, D. L. (2009). Cognitive-behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*, *77*, 595–606. http://doi.org/10.1037/a00116032

Tibshirani, R. (1996). Regression shrinkage and selection via the lasso. *Journal of the Royal Statistical Society. Series B (Methodological)*, 267–288.

Wichers, M. (2014). The dynamic nature of depression: a new micro-level perspective of mental disorder that meets current challenges. *Psychological Medicine*, *6160*, 1–12. http://doi.org/10.1017/S0033291713001979

Wickham, H. (2016). *ggplot2: Elegant Graphics for Data Analysis*. New York, NY: Springer-Verlag New York.

Wickham, H., François, R., Henry, L., & Müller, K. (2019). dplyr: A grammar of data manipulation. R package version 0.8.0.1. https://cran.r-project.org/package=dplyr

World Health Organization (1992). *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines* (10th Ed.). Geneva, Switzerland: World Health Organization.

Yang, B. (2005). Factor Analysis Methods. In R. A. Swanson & E. F. I. Holton (Eds.), *Research in Organizations: Foundations and Methods of Inquiry* (pp. 181–199). San Francisco, CA: Berrett-Koehler Publishers, Inc.

Yeung, R. R. (1996). The acute effects of exercise on mood state. *Journal of Psychosomatic Research*, *40*, 123–141. http://doi.org/https://doi-org.libproxy.wustl.edu/10.1016/0022-3999(95)00554-4

Zimmerman, J., Woods, W. C., Ritter, S., Happel, M., Masuhr, O., Jaeger, U., & Spitzer, C. (2018). Integrating structure and dynamics in personality assessment: First steps toward the development and validation of a Personality Dynamics Diary. *Psychological Assessment*. http://doi.org/10.31234/osf.io/5zcth

Appendix A.

Please complete the following questions based on how you were feeling when you received the notification.

1) How down do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

2) How happy do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

3) How calm do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

4) How irritated do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

5) How anxious do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

6) How lonely do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

7) How accomplished do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

8) How hungry do you feel right now?

0 1 2 3 4 5 6 7 8 9 10

9) How physically active (e.g., walking, exercising, stretching) have you been since the last beep?

0 1 2 3 4 5 6 7 8 9 10

10) Are you currently avoiding a social situation or interaction?

0 1 2 3 4 5 6 7 8 9 10

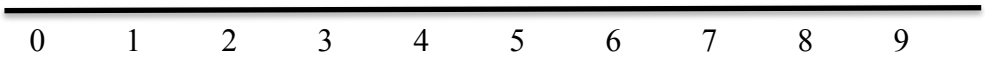
11) How drowsy are you right now?

0 1 2 3 4 5 6 7 8 9 10

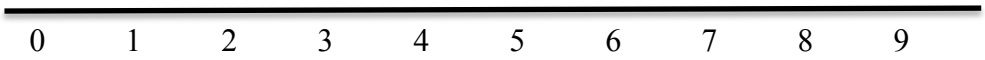
12) How pleased are you with your experience right now?

0 1 2 3 4 5 6 7 8 9 10

13) How restless do you feel right now?



14) How focused are you right now?



Appendix B.

Sample code for GVAR analyses using R:

```
library(dplyr)
library(ggplot2)
library(stats)
library(modelr)
library(graphicalVAR)

#Read in data
MyMood_001 <- read.csv("MyMood_001.csv")
MyMood_001 <- as.data.frame(MyMood_001)

MyMood_001$Day <- as.numeric(MyMood_001$Day)
MyMood_001$Survey <- as.numeric(MyMood_001$Survey)
MyMood_001$Down <- as.numeric(MyMood_001$Down)
MyMood_001$Happy <- as.numeric(MyMood_001$Happy)
MyMood_001$Calm <- as.numeric(MyMood_001$Calm)
MyMood_001$Irritated <- as.numeric(MyMood_001$Irritated)
MyMood_001$Anxious <- as.numeric(MyMood_001$Anxious)
MyMood_001$Lonely <- as.numeric(MyMood_001$Lonely)
MyMood_001$Accomplished <- as.numeric(MyMood_001$Accomplished)
MyMood_001$Hungry <- as.numeric(MyMood_001$Hungry)
MyMood_001$PhysAct <- as.numeric(MyMood_001$PhysAct)
MyMood_001$SocAvoid <- as.numeric(MyMood_001$SocAvoid)
MyMood_001$Drowsy <- as.numeric(MyMood_001$Drowsy)
MyMood_001$Pleased <- as.numeric(MyMood_001$Pleased)
MyMood_001$Restless <- as.numeric(MyMood_001$Restless)
MyMood_001$Focused <- as.numeric(MyMood_001$Focused)

descr(MyMood_001) #Examine range of items

CorTable <- cor(MyMood_001, use = "pairwise.complete.obs")
round(CorTable, 2) #Are there time effects?

#Code for creating residuals, if there are significant time effects
MyMood_001Reg <- MyMood_001 %>% gather(key = item, value = value, Down:Focused)
 %>%
  group_by(item) %>%
  nest() %>%
  mutate(model = map(data, ~lm(value ~ Day, data = .)),
         residuals = map2(data, model, add_residuals)) %>%
  unnest(residuals) %>%
```

```
  select(-value) %>%
  spread(key = item, value = resid)

CorTable <- cor(MyMood_001Reg, use = "pairwise.complete.obs")
round(CorTable, 2) #Are certain ISM items highly correlated?

#Code for composting highly-correlated items if necessary
MyMood_001RegComp <- transmute(MyMood_001Reg, Day, Survey, Down, HapPls =
 (Pleased + Happy)/2, Calm, Irritated, Anxious, Accomplished, Lonely, Hungry, PhysAct,
 SocAvoid, Drowsy, Restless, Focused)

#Code for GVAR model using a composited dataset
MyMood_001RegCompGVAR <- graphicalVAR(MyMood_001RegComp, gamma = .1, verbose
 = FALSE, dayvar = c("Day"), beepvar = c("Survey"), vars = c("Down", "Calm", "Irritated",
 "Anxious", "Accomplished", "Lonely", "Hungry", "PhysAct", "SocAvoid", "Drowsy",
 "HapPls", "Restless", "Focused"))
plot(MyMood_001RegCompGVAR)

#Code for GVAR model using the non-composited dataset
MyMood_001RegGVAR <- graphicalVAR(MyMood_001Reg, gamma = .1, verbose = FALSE,
 dayvar = c("Day"), beepvar = c("Survey"), vars = c("Down", "Happy", "Calm", "Irritated",
 "Anxious", "Accomplished", "Lonely", "Hungry", "PhysAct", "SocAvoid", "Drowsy",
 "Pleased", "Restless", "Focused"))
plot(MyMood_001RegGVAR)
```

Code for Lag 1 idiographic DSEM analyses using Mplus:

```
INPUT INSTRUCTIONS
TITLE:     DSEM for MyMood_001: Down only
DATA:      FILE IS MyMood_001mplusDSEM.csv;
VARIABLE:  NAMES ARE Day Surv Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct
SocAv Drow Pleas Restl Foc;
USEVARIABLES ARE Day Surv Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv
Drow Pleas Restl Foc;
lagged = Down(1) Hap(1) Calm(1) Irrit(1) Anx(1) Lon(1) Accomp(1) Hung(1) PhyAct(1)
SocAv(1) Drow(1) Pleas(1) Restl(1) Foc(1);
MISSING = ALL (-99);
ANALYSIS: ESTIMATOR = BAYES;
PROCESSORS = 2;
fbiter = (20800);
!thin = 100;

model:
Down on Surv;
Hap on Surv;
```

```
Calm on Surv;
Irrit on Surv;
Anx on Surv;
Lon on Surv;
Accomp on Surv;
Hung on Surv;
PhyAct on Surv;
SocAv on Surv;
Drow on Surv;
Pleas on Surv;
Restl on Surv;
Foc on Surv;

Down on Day;
Hap on Day;
Calm on Day;
Irrit on Day;
Anx on Day;
Lon on Day;
Accomp on Day;
Hung on Day;
PhyAct on Day;
SocAv on Day;
Drow on Day;
Pleas on Day;
Restl on Day;
Foc on Day;

Down on Down&1 Hap&1 Calm&1 Irrit&1 Anx&1 Lon&1 Accomp&1 Hung&1
PhyAct&1 SocAv&1 Drow&1 Pleas&1 Restl&1 Foc&1;

OUTPUT: TECH1 TECH8;
stand;
PLOT: TYPE = PLOT3;
```

Code for Lag 2 idiographic DSEM analyses using Mplus:

```
INPUT INSTRUCTIONS
TITLE:     DSEM for MyMood_003: Down only
DATA:      FILE IS MyMood_003mplusDSEM.csv;
VARIABLE:  NAMES ARE Day Surv Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct
SocAv Drow Pleas Restl Foc;
USEVARIABLES ARE Day Surv Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv
Drow Pleas Restl Foc;
lagged = Down(2) Hap(2) Calm(2) Irrit(2) Anx(2) Lon(2) Accomp(2) Hung(2) PhyAct(2)
SocAv(2) Drow(2) Pleas(2) Restl(2) Foc(2);
```

```
MISSING = ALL (-99);
ANALYSIS: ESTIMATOR = BAYES;
PROCESSORS = 2;
fbiter = (10400);
!thin = 100;

model:
Down on Surv;
Hap on Surv;
Calm on Surv;
Irrit on Surv;
Anx on Surv;
Lon on Surv;
Accomp on Surv;
Hung on Surv;
PhyAct on Surv;
SocAv on Surv;
Drow on Surv;
Pleas on Surv;
Restl on Surv;
Foc on Surv;

Down on Day;
Hap on Day;
Calm on Day;
Irrit on Day;
Anx on Day;
Lon on Day;
Accomp on Day;
Hung on Day;
PhyAct on Day;
SocAv on Day;
Drow on Day;
Pleas on Day;
Restl on Day;
Foc on Day;

Down on Down&1 Hap&1 Calm&1 Irrit&1 Anx&1 Lon&1 Accomp&1 Hung&1
PhyAct&1 SocAv&1 Drow&1 Pleas&1 Restl&1 Foc&1;

Down on Down&2 Hap&2 Calm&2 Irrit&2 Anx&2 Lon&2 Accomp&2 Hung&2
PhyAct&2 SocAv&2 Drow&2 Pleas&2 Restl&2 Foc&2;

OUTPUT: TECH1 TECH8;
stand;
PLOT: TYPE = PLOT3;
```

Code for Lag 1 ML-DSEM analyses using Mplus:

```
INPUT INSTRUCTIONS
TITLE: MyMood Multilevel Model - Down variable only
DATA: file is MyMood_datafull_DSEM.csv;
VARIABLE:
NAMES      = ID Day Surv No Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv
Drow Pleas Restl Foc;
CLUSTER    = ID;
USEVAR     = Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv Drow Pleas Restl
Foc Day Surv;
LAGGED     = Down(1) Hap(1) Calm(1) Irrit(1) Anx(1) Lon(1) Accomp(1) Hung(1) PhyAct(1)
SocAv(1) Drow(1) Pleas(1) Restl(1) Foc(1);
TINTERVAL  = No(1);
MISSING    = all(-99);
ANALYSIS:  TYPE IS TWOLEVEL RANDOM;
ESTIMATOR = BAYES;
PROC = 2;

MODEL:
 %WITHIN%
!Random slope for Down_t regressed on Down_t-1 (autoregression), etc.
p_Dwn | Down ON Down&1;

!Random slope for Hap_t-1, etc. predicting Down_t (cross-lagged)
p_HapD | Down ON Hap&1;
p_ClmD | Down ON Calm&1;
p_IrrD | Down ON Irrit&1;
p_AnxD | Down ON Anx&1;
p_LonD | Down ON Lon&1;
p_AccD | Down ON Accomp&1;
p_HngD | Down ON Hung&1;
p_PhAD | Down ON PhyAct&1;
p_SAvD | Down ON SocAv&1;
p_DrwD | Down ON Drow&1;
p_PlsD | Down ON Pleas&1;
p_RstD | Down ON Restl&1;
p_FocD | Down ON Foc&1;

!Random slope for Down_t regressed on Day, etc.
Day_Dwn | Down ON Day;
Day_Hap | Hap ON Day;
Day_Clm | Calm ON Day;
Day_Irr | Irrit ON Day;
Day_Anx | Anx ON Day;
```

```
Day_Lon | Lon ON Day;
Day_Acc | Accomp ON Day;
Day_Hng | Hung ON Day;
Day_PhA | PhyAct ON Day;
Day_SAv | SocAv ON Day;
Day_Drw | Drow ON Day;
Day_Pls | Pleas ON Day;
Day_Rst | Restl ON Day;
Day_Foc | Foc ON Day;

!Random slope for Down_t regressed on Surv, etc.
Surv_Dwn | Down ON Surv;
Surv_Hap | Hap ON Surv;
Surv_Clm | Calm ON Surv;
Surv_Irr | Irrit ON Surv;
Surv_Anx | Anx ON Surv;
Surv_Lon | Lon ON Surv;
Surv_Acc | Accomp ON Surv;
Surv_Hng | Hung ON Surv;
Surv_PhA | PhyAct ON Surv;
Surv_SAv | SocAv ON Surv;
Surv_Drw | Drow ON Surv;
Surv_Pls | Pleas ON Surv;
Surv_Rst | Restl ON Surv;
Surv_Foc | Foc ON Surv;

!Random unique innovation variance (error)
errD | Down;
errH | Hap;
errC | Calm;
errI | Irrit;
errA | Anx;
errL | Lon;
errAc | Accomp;
errHn | Hung;
errPA | PhyAct;
errSA | SocAv;
errDr | Drow;
errP | Pleas;
errR | Restl;
errF | Foc;

%BETWEEN%
p_Dwn;
p_HapD;
p_ClmD;
```

```
p_IrrD;
p_AnxD;
p_LonD;
p_AccD;
p_HngD;
p_PhAD;
p_SAvD;
p_DrwD;
p_PlsD;
p_RstD;
p_FocD;

OUTPUT: TECH1 TECH8 STDYX STAND(CLUSTER) FSCOMPARISON;
PLOT: TYPE = PLOT3; FACTOR =ALL;
savedata: file is ests2-21-19.dat;
save = fscores (1000);
missflag=-99;
```

Code for Lag 2 ML-DSEM analyses using Mplus:

```
INPUT INSTRUCTIONS
TITLE: MyMood Multilevel Model - Down variable only, lag 2
DATA: file is MyMood_datafull_DSEM.csv;
VARIABLE:
NAMES      = ID Day Surv No Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv
Drow Pleas Restl Foc;
CLUSTER    = ID;
USEVAR     = Down Hap Calm Irrit Anx Lon Accomp Hung PhyAct SocAv Drow Pleas Restl
Foc Day Surv;
LAGGED     = Down(2) Hap(2) Calm(2) Irrit(2) Anx(2) Lon(2) Accomp(2) Hung(2) PhyAct(2)
SocAv(2) Drow(2) Pleas(2) Restl(2) Foc(2);
TINTERVAL  = No(2);
MISSING    = all(-99);
ANALYSIS:  TYPE IS TWOLEVEL RANDOM;
ESTIMATOR = BAYES;
PROC = 2;

MODEL:
 %WITHIN%
!Random slope for Down_t regressed on Down_t-1 (autoregression), etc.
p_Dwn | Down ON Down&1;
p_Dwn2 | Down ON Down&2;

!Random slope for Hap_t-1, etc. predicting Down_t (cross-lagged)
p_HapD | Down ON Hap&1;
p_ClmD | Down ON Calm&1;
```

```
p_IrrD | Down ON Irrit&1;
p_AnxD | Down ON Anx&1;
p_LonD | Down ON Lon&1;
p_AccD | Down ON Accomp&1;
p_HngD | Down ON Hung&1;
p_PhAD | Down ON PhyAct&1;
p_SAvD | Down ON SocAv&1;
p_DrwD | Down ON Drow&1;
p_PlsD | Down ON Pleas&1;
p_RstD | Down ON Restl&1;
p_FocD | Down ON Foc&1;

p_HapD2 | Down ON Hap&2;
p_ClmD2 | Down ON Calm&2;
p_IrrD2 | Down ON Irrit&2;
p_AnxD2 | Down ON Anx&2;
p_LonD2 | Down ON Lon&2;
p_AccD2 | Down ON Accomp&2;
p_HngD2 | Down ON Hung&2;
p_PhAD2 | Down ON PhyAct&2;
p_SAvD2 | Down ON SocAv&2;
p_DrwD2 | Down ON Drow&2;
p_PlsD2 | Down ON Pleas&2;
p_RstD2 | Down ON Restl&2;
p_FocD2 | Down ON Foc&2;

!Random slope for Down_t regressed on Day, etc.
Day_Dwn | Down ON Day;
Day_Hap | Hap ON Day;
Day_Clm | Calm ON Day;
Day_Irr | Irrit ON Day;
Day_Anx | Anx ON Day;
Day_Lon | Lon ON Day;
Day_Acc | Accomp ON Day;
Day_Hng | Hung ON Day;
Day_PhA | PhyAct ON Day;
Day_SAv | SocAv ON Day;
Day_Drw | Drow ON Day;
Day_Pls | Pleas ON Day;
Day_Rst | Restl ON Day;
Day_Foc | Foc ON Day;

!Random slope for Down_t regressed on Surv, etc.
Surv_Dwn | Down ON Surv;
Surv_Hap | Hap ON Surv;
Surv_Clm | Calm ON Surv;
```

```
Surv_Irr | Irrit ON Surv;
Surv_Anx | Anx ON Surv;
Surv_Lon | Lon ON Surv;
Surv_Acc | Accomp ON Surv;
Surv_Hng | Hung ON Surv;
Surv_PhA | PhyAct ON Surv;
Surv_SAv | SocAv ON Surv;
Surv_Drw | Drow ON Surv;
Surv_Pls | Pleas ON Surv;
Surv_Rst | Restl ON Surv;
Surv_Foc | Foc ON Surv;

!Random unique innovation variance (error)
errD | Down;
errH | Hap;
errC | Calm;
errI | Irrit;
errA | Anx;
errL | Lon;
errAc | Accomp;
errHn | Hung;
errPA | PhyAct;
errSA | SocAv;
errDr | Drow;
errP | Pleas;
errR | Restl;
errF | Foc;

%BETWEEN%
p_Dwn;
p_HapD;
p_ClmD;
p_IrrD;
p_AnxD;
p_LonD;
p_AccD;
p_HngD;
p_PhAD;
p_SAvD;
p_DrwD;
p_PlsD;
p_RstD;
p_FocD;

p_Dwn2;
p_HapD2;
```

```
p_ClmD2;
p_IrrD2;
p_AnxD2;
p_LonD2;
p_AccD2;
p_HngD2;
p_PhAD2;
p_SAvD2;
p_DrwD2;
p_PlsD2;
p_RstD2;
p_FocD2;

OUTPUT: TECH1 TECH8 STDYX STAND(CLUSTER) FSCOMPARISON;
PLOT: TYPE = PLOT3; FACTOR =ALL;
savedata: file is estslagtwo2-25-19.dat;
save = fscores (1000);
missflag=-99;
```

Code for Figure 4 (visualizing overlap between idiographic and ML models) using R:

```
library(MplusAutomation) #reading in individual-level estimates generated from the ML model
MLEstimates <- getSavedata_Data("multilevel_4.0withtwiceasmanyitsandestimates.out")
MLEstimates <- readModels("multilevel_4.0withtwiceasmanyitsandestimates Estimates <-
 read.csv("Lag1Estimates - MLID.csv")
Estimates <- as.data.frame(Estimates).out", what="savedata")
write.csv(MLEstimates, file = "ML_1_Estimates.csv")

#Identifying participants with low numbers of observations
Estimates$ID2 <- NA
Estimates$ID2 <- ifelse(Estimates$ID == 1, 1, NA)
Estimates$ID2 <- ifelse(Estimates$ID == 4, 4, Estimates$ID2)
Estimates$ID2 <- ifelse(Estimates$ID == 7, 7, Estimates$ID2)
Estimates$ID2 <- ifelse(Estimates$ID == 8, 8, Estimates$ID2)

Estimates %>%
gather(key = var, value = value, ML_DWN.Md, ID_DWN.Mn) %>%
ggplot(aes(y = value, x = 1, label = ID2, fill = var, color = var)) +
  geom_violin(alpha = .3, trim = FALSE, position = position_dodge(0)) +
  geom_point(size = 0, alpha = .8) +
    geom_text_repel(
    nudge_x      = 0.25,
    direction    = "y",
    hjust        = 0,
    segment.size = 0.5,
    size = 6
```

```
  ) +
coord_flip() +
scale_fill_manual(values=c("gray37", "gray5")) +
scale_color_manual(values=c("gray37", "gray5")) +
labs(title="TITLE", y="Estimate", x = "Var") +
theme(plot.title=element_text(hjust=0.5), legend.position = "none") +
theme_classic() +
theme(
  plot.title   = element_text(hjust = 0.5),
  legend.position = "none",
  axis.ticks.y = element_blank(),
  axis.text.y  = element_blank()
) +
xlim(1, 1.3)
```

Table 1. Estimates from the Lag 1 multilevel model.

Path	Estimate	Posterior S.D.	95% Credible interval, Lower bound	95% Credible interval, Upper bound
Day	0.04*	0.02	0.01	0.07
Survey	0.02	0.03	-0.04	0.08
Down &1	0.18*	0.02	0.13	0.23
Happy &1	-0.04	0.07	-0.09	0.01
Calm &1	-0.04*	0.02	-0.08	0.00
Irritable &1	0.02	0.02	-0.02	0.06
Anxious &1	0.04	0.02	-0.01	0.08
Lonely &1	0.06*	0.02	0.02	0.11
Accomplished &1	-0.02	0.02	-0.07	0.02
Hungry &1	-0.02	0.02	-0.05	0.02
Physical Activity &1	0.04*	0.02	0.002	0.07
Social Avoidance &1	0.04*	0.02	0.01	0.08
Drowsy &1	0.01	0.02	-0.03	0.05
Pleased &1	-0.002	0.03	-0.05	0.05
Restless &1	0.01	0.02	-0.03	0.05
Focused &1	-0.003	0.02	-0.04	0.04

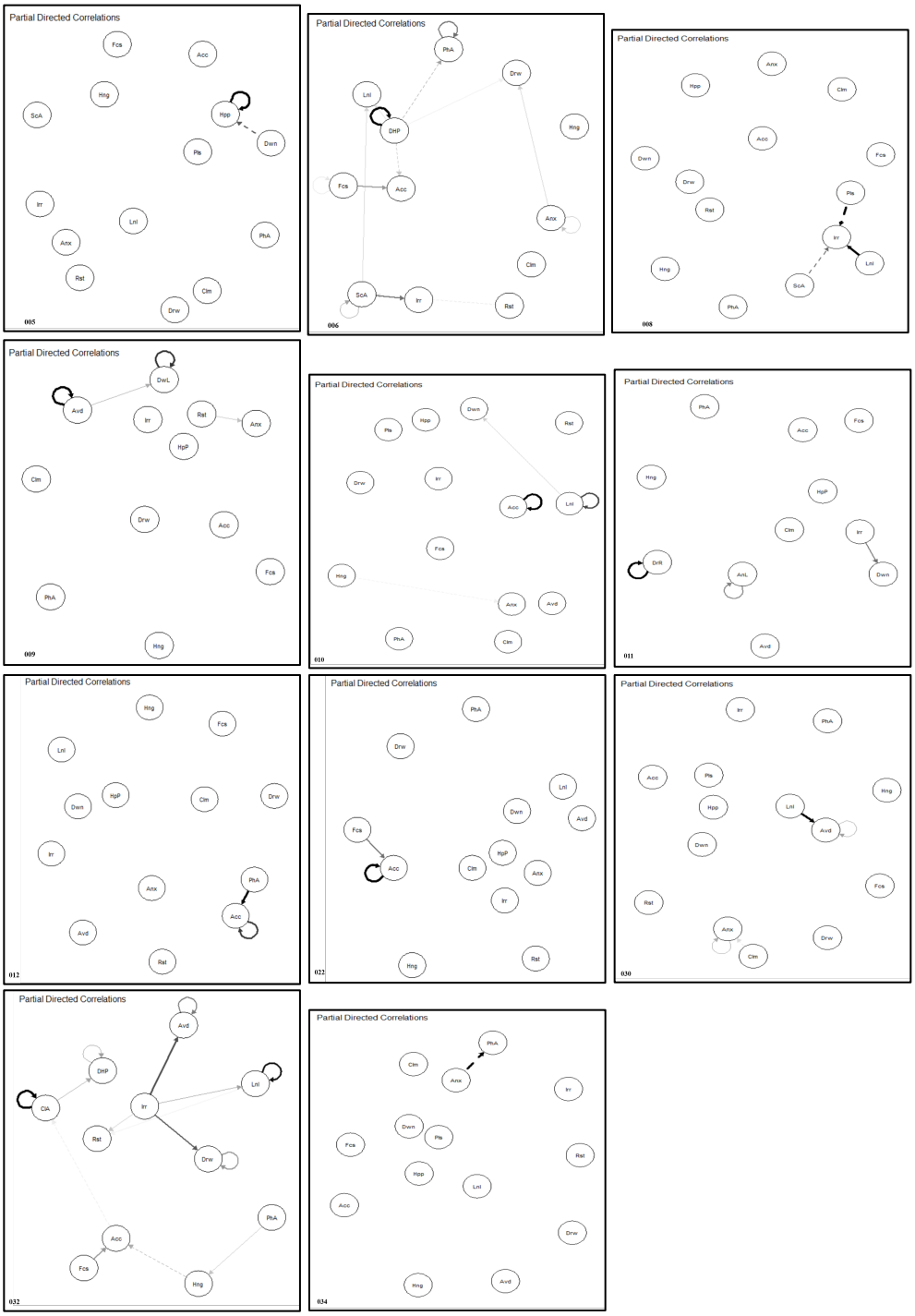
Note. * denotes a 95% credible interval that does not include 0, suggesting statistical significance. &1 refers to Lag 1 variables.

Table 2. Estimates from the Lag 2 multilevel model.

Path	Estimate	Posterior S.D.	95% Credible interval, Lower bound	95% Credible interval, Upper bound
Day	0.03	0.02	-0.01	0.06
Survey	0.02	0.03	-0.04	0.09
Down &1	0.18*	0.02	0.13	0.22
Happy &1	-0.04	0.02	-0.09	0.01
Calm &1	-0.05*	0.02	-0.08	-0.002
Irritable &1	0.01	0.02	-0.03	0.05
Anxious &1	0.03	0.02	-0.01	0.07
Lonely &1	0.05*	0.02	0.01	0.09
Accomplished &1	-0.03	0.02	-0.07	0.02
Hungry &1	-0.02	0.02	-0.05	0.01
Physical activity &1	0.03	0.02	-0.01	0.07
Social avoidance &1	0.03	0.02	-0.01	0.07
Drowsy &1	0.000	0.02	-0.04	0.04
Pleased &1	0.005	0.02	-0.04	0.05
Restless &1	0.0001	0.02	-0.04	0.04
Focused &1	0.001	0.02	-0.04	0.04
Down &2	0.08*	0.03	0.04	0.14
Happy &2	0.004	0.03	-0.05	0.06
Calm &2	-0.01	0.03	-0.06	0.04
Irritable &2	0.02	0.02	-0.03	0.07
Anxious &2	0.03	0.03	-0.02	0.08
Lonely &2	0.02	0.02	-0.03	0.06
Accomplished &2	-0.02	0.03	-0.06	0.04
Hungry &2	0.03	0.02	-0.003	0.07
Physical activity &2	0.03	0.02	-0.01	0.07
Social avoidance &2	0.003	0.02	-0.04	0.05
Drowsy &2	0.03	0.02	-0.01	0.08
Pleased &2	0.04	0.03	-0.02	0.10
Restless &2	0.04	0.02	-0.01	0.08
Focused &2	0.03	0.02	-0.01	0.07

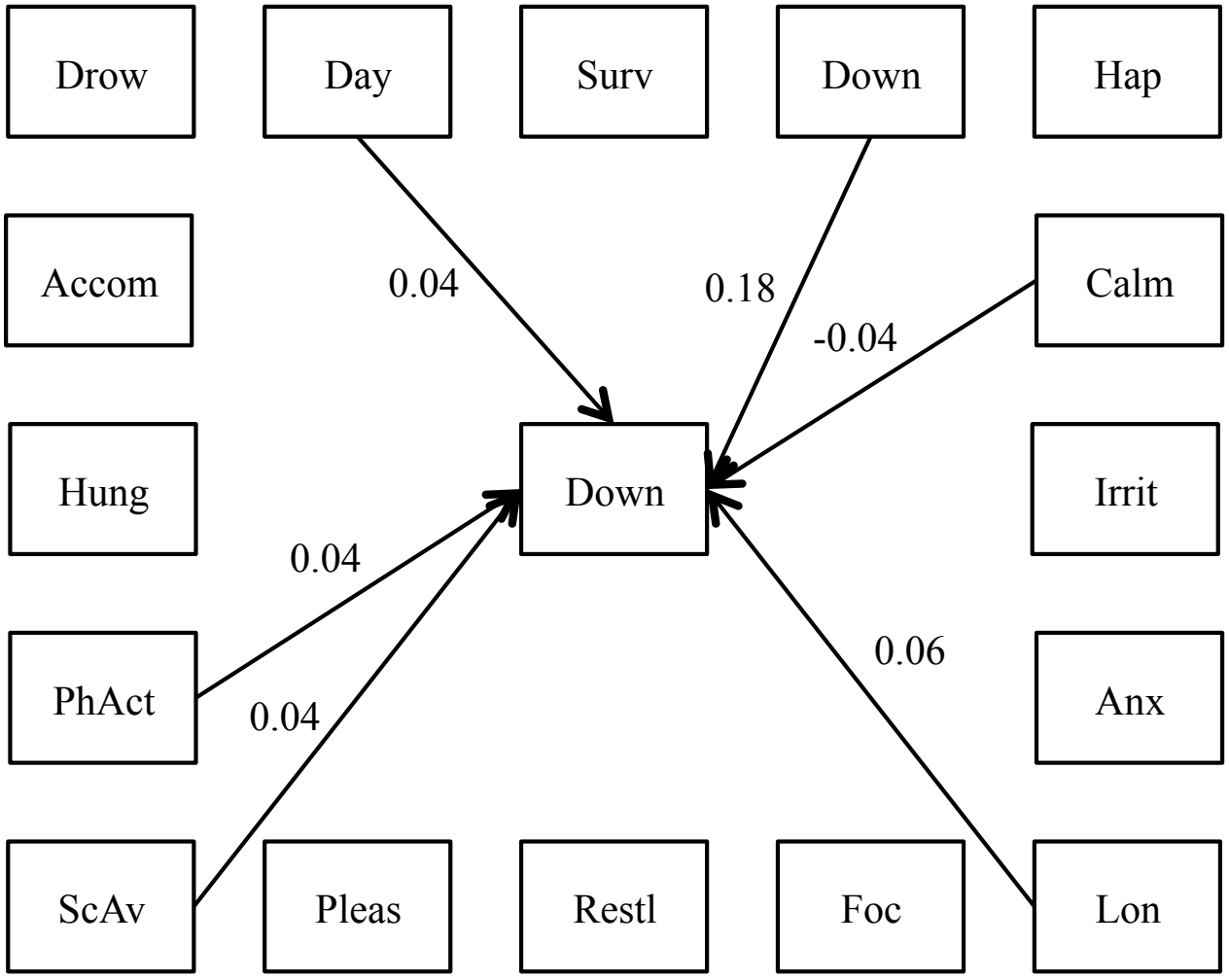
Note. * denotes a 95% credible interval that does not include 0, suggesting statistical significance. &1 refers to Lag 1 variable.

Figure 1. Directed effects from non-composited and composited GVAR models.



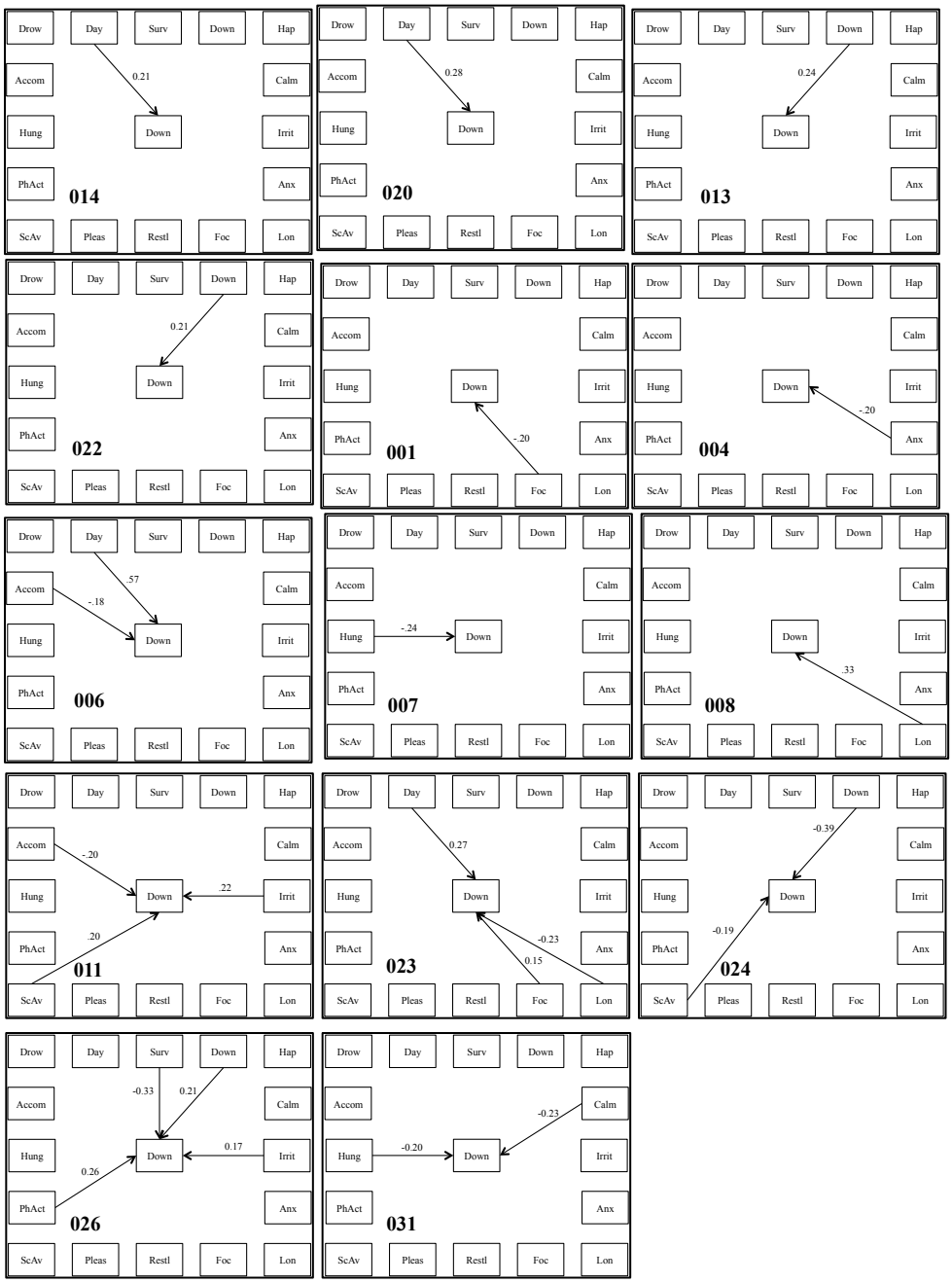
Note. Dashed lines indicate a negative relationship; solid lines indicate a positive relationship. Loops indicate an autoregressive relationship. The saturation of the line corresponds to the magnitude of the relationship (darker lines indicate a stronger relationship). Bolded numbers refer to participant ID. Participants with IDs 5, 8, 10, 30, and 34 did not have any ISM items with correlations > .65 so their non-composited models are displayed here. Participants with IDs 6, 9, 11, 12 22, and 32 had ISM items with correlations > .65 so their composited models are displayed here. Clm = Calm; Irr = Irritable; Lnl = Lonely; Hng = Hungry; Pls = Pleased; Acc = Accomplished; Hpp = Happy; Avd = Social avoidance; Rst = Restless; Anx = Anxious; Dwn = Down; PhA = Physical activity; Fcs = Focused; Drw = Drowsy; DHP = Down, Happy (reverse-scored), Pleased (reverse-scored); HpP = Happy, Pleased; ClA = Calm (reverse-scored), Anxious; DrR = Drowsy, Restless; AnL = Anxious, Lonely.

Figure 2. Multilevel model demonstrating statistically significant Lag 1 pathways.



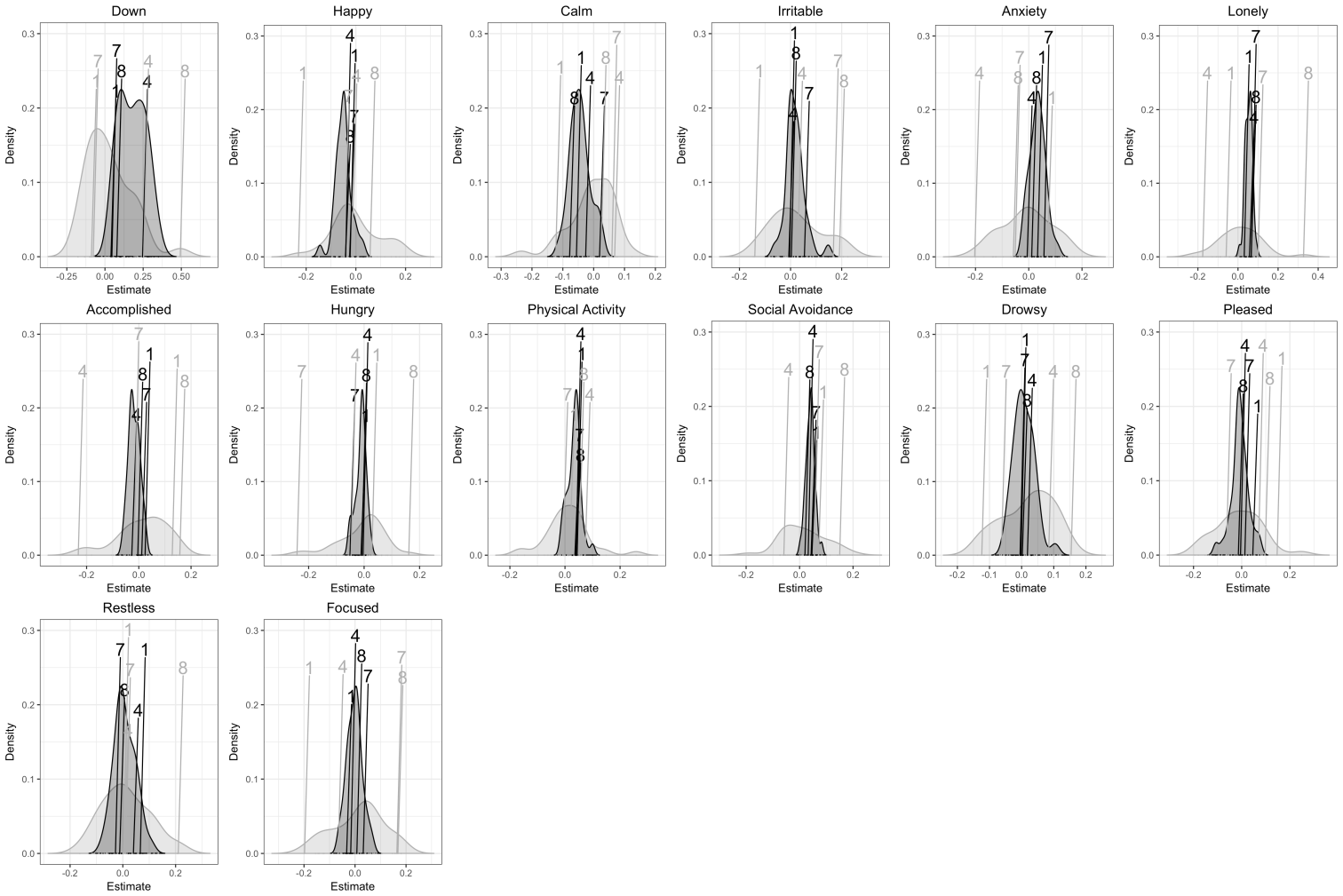
Note. Only standardized pathways with credible intervals that do not include 0 are demonstrated here. Hap = Happy; Irrit = Irritable; Anx = Anxious; Lon = Lonely; Foc = Focused; Restl = Restless; Pleas = Pleased; ScAv = Social avoidance; PhAct = Physical activity; Hung = Hungry; Accom = Accomplished; Drow = Drowsy; Surv = Survey

Figure 3. Idiographic models demonstrating statistically significant Lag 1 pathways



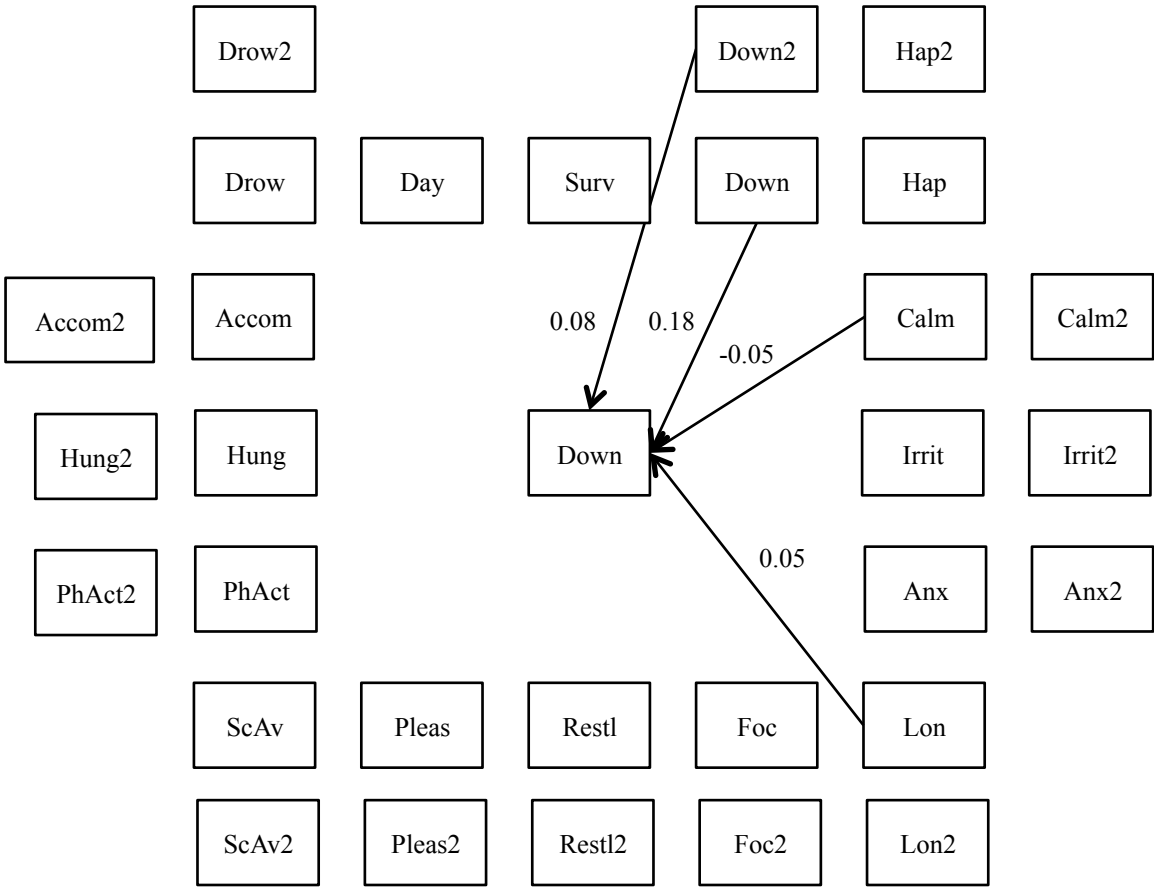
Note. Only standardized pathways with credible intervals that do not include 0 (indicating statistical significance) are shown here. Bolded numbers refer to participant ID. Surv = Survey; Hap = Happy; Irrit = Irritable; Anx = Anxious; Lon = Lonely; Foc = Focused; Restl = Restless; Pleas = Pleased; ScAv = Social avoidance; PhAct = Physical Activity; Hung = Hungry; Accom = Accomplishment; Drow = Drowsy

Figure 4. Overlap between multilevel and idiographic estimates.



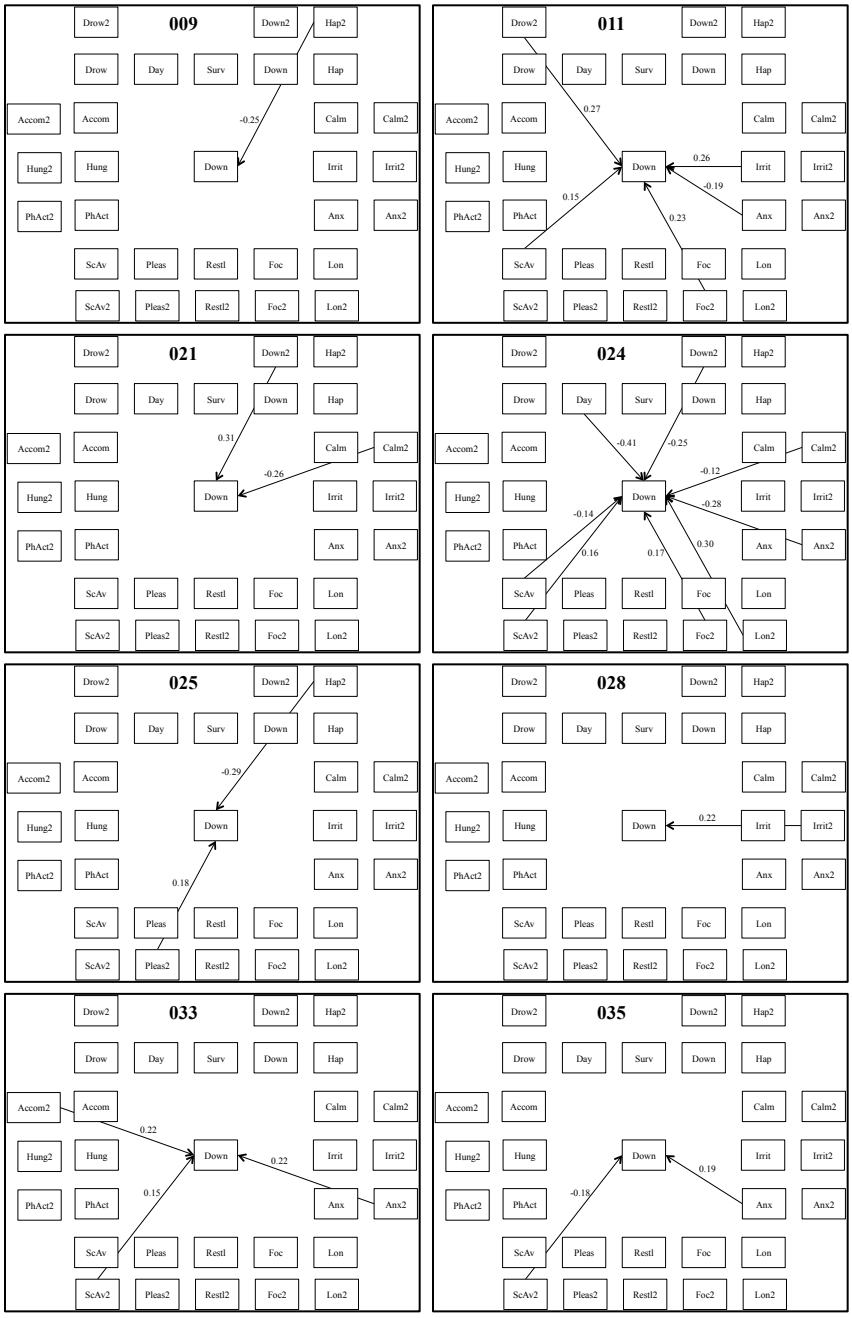
Note. The light grey density plot represents the individual-level estimates for each ISM pathway predicting down from the idiographic DSEM. The dark grey density plot represents the individual-level estimates for each ISM pathway predicting down from the ML-DSEM. Participants with fewer than 100 time points (IDs 1, 4, 7, and 8) are noted.

Figure 5. Multilevel model demonstrating statistically significant Lag 2 pathways.



Note. Only standardized pathways with credible intervals that do not include 0 are demonstrated here. Hap = Happy; Irri
Irritable; Anx = Anxious; Lon = Lonely; Foc = Focused; Restl = Restless; Pleas = Pleased; ScAv = Social avoidance; PhA
Physical activity; Hung = Hungry; Accom = Accomplished; Drow = Drowsy; Surv = Survey

Figure 6. Idiographic models demonstrating statistically significant Lag 2 pathways.



Note. Only models with significant Lag 2 cross-lagged paths predicting down in addition to Lag 2 autoregressive effects are shown here. There was one additional participant whose Lag 2 model included only a Lag 2 autoregressive effect. Only standardized pathways with credible intervals that do not include 0 (indicating statistical significance) are shown here. Bolded numbers refer to participant ID. Day = Day; Surv = Survey; Down = Down; Hap = Happy; Irrit = Irritable; Anx = Anxious; Lon = Lonely; Foc = Focused; Restl = Restless; Pleas = Pleased; ScAv = Social avoidance; PhAct = Physical Activity; Hung = Hungry; Accom = Accomplishment; Drow = Drowsy. Down2 = Down, Lag 2; Hap2 = Happy, Lag 2; Irrit2 = Irritable, Lag 2; Anx2 = Anxious, Lag 2; Lon2 = Lonely, Lag 2; Foc2 = Focused, Lag 2; Restl2 = Restless, Lag 2; Pleas2 = Pleased, Lag 2; ScAv2 = Social avoidance, Lag 2; PhAct2 = Physical activity, Lag 2; Hung2 = Hungry, Lag 2; Accom2 = Accomplished, Lag 2; Drow2 = Drowsy, Lag 2.